

# South African Medical Journal

## Suid-Afrikaanse Tydskrif vir Geneeskunde

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Cape Town, 21 July 1956  
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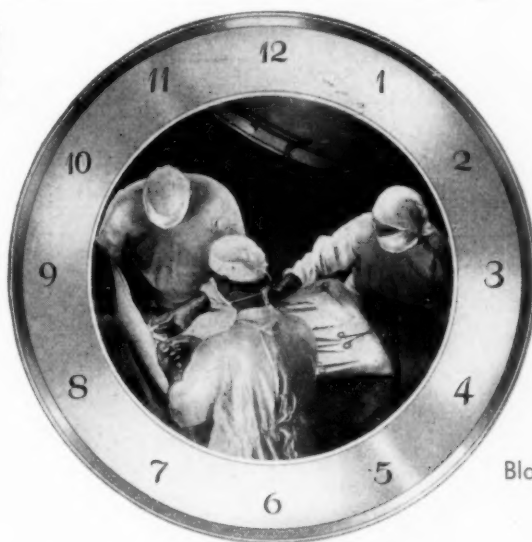
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### VAN DIE REDAKSIE

#### DIE BESTRYDING VAN KAAKKLEM

Kaakklem val vandag nog eweveel binne die bestek van die epidemiologie as van die kliniekwerk. Totdat aktiewe bestandmaking op die hele bevolking toegepas word<sup>1</sup> sal kaakklem voortgaan om sy jaarlikse aantal slagoffers te eis. Die hoogste sterftesyfer kom onder adolessente voor; hulle kom tewens baie meer met besmette grond in aanraking as ander ouderdomsgroepe. In Brittanje, waar die epidemiologiese standaard betreklik hoog is, sterf gemiddeld 50 mense elke jaar aan kaakklem.<sup>2</sup> Die Noord-Amerikaners het baanbrekerswerk gedoen insake die voorbehoedende gebruik van kaakklem-toksoied om aktiewe onvatbaarheid te verleen, met die gevolg dat daar in die laaste oorlog slegs een geval van kaakklem onder al die slagveld-gewondes van die Amerikaanse leër voorgekom het. Soortgelyke resultate, wel minder skouspelagtig, is ook in Brittanje behaal. Twee dosisse, 6 weke uit mekaar, word aanbeveel, met 'n derde 6 maande later. By kinders kan dit saam met die difterie-inenting gedoen word; dit is vandag verpligtend in Denemarke en Frankryk<sup>3</sup> en gebruikelik in die VSA en Kanada. Opwekkingsdosisse al om die 4 jaar word aanbeveel. Al die Amerikaanse gewondes het so 'n opwekkingsdosis gekry in plaas van die kaakklem teengif wat gewoonlik gebruik word en wat op sy beste die pasiënt maar twee weke lank passief onvatbaar maak. Gewoontes word maar seker net so langsaam gebore as wat hulle uitsterf, sodat dit miskien 'n tyd lank sal duur voordat aktiewe bestandmaking teen kaakklem as reël in Suid-Afrika toegepas sal word. Nogtans het die ondervinding geleer dat die velbesering waardeur kaakklem kan ontstaan dikwels so gering is dat dit verontagsaam word totdat die besmetting intree.

Intussen vind daar fundamentele her-onderzoek en verandering plaas op die gebied van die gebruikelike begrippe oor die beheer van die akute kaakklem-aanval. Dit word beweer<sup>4</sup> dat die werking van die kaakklem-toksien op die onderdrukking van die remsprake op die senuweetussenselle berus. Hierdie verwydering van fisiologiese stremming stel die sinaptiese verbindings bloot aan al die aangevoerde opwekkende prikkels, en selfs geringe prikkels word in aktiewe

### EDITORIAL

#### COMBATING TETANUS

Tetanus remains as much the field of the epidemiologist as of the clinician. Until active immunization is applied to the whole population,<sup>1</sup> tetanus will continue to take its annual toll of lives. The highest death rate occurs amongst adolescents, whose contact with infected soil is likely to be greater than that of people in other age-groups. In Britain, where epidemiological standards are fairly high, about 50 persons die each year of tetanus.<sup>2</sup> The North Americans have pioneered the prophylactic use of tetanus toxoid to induce active immunity, and as a result only one case of tetanus occurred among all the battle casualties in the American army during the last war. Similar, though less spectacular, results were achieved by the British. Two doses are advised, spaced 6 weeks apart, followed by a third dose 6 months later. In children this may be combined with diphtheria immunization, as is now compulsory in Denmark and France,<sup>3</sup> and widely practised in the United States and Canada. Booster doses every 4 years are advised. All American casualties were given a booster dose instead of the tetanus antitoxin customarily employed, which at best invests the patient with only 2 weeks of passive immunity. Customs are likely to take as long to be born as to die, so that one cannot anticipate the introduction of active immunization against tetanus in South Africa for some time. Nevertheless, experience has shown time and again that the offending skin-lesion in tetanus is often so trivial as to be ignored until the infection sets in.

Meanwhile, traditional concepts on the management of the acute attack of tetanus are undergoing fundamental re-examination and change. The tetanus toxin acts, it is said,<sup>4</sup> by depressing inhibitory stimuli on the interneurons of the nervous system. This removal of physiological restraint leaves the synaptic junctions

teit uitgedruk. Tensy die afvoerende senuweeëtketting onderbreek word, of die pasiënt met kalmeermiddels fisiologies op normaal gebring word, veroorsaak hierdie abnormale beweeglikheid spoedig uitputting en die dood. Twee soorte kliniese middels is reeds gebruik. Die radikaalste metode is om totale verlamming van die hele willekeurige spierstelsel te veroorsaak met 'n spier-senuwee-blokkeringsmiddel soos kurare. Om die emosionele afgrysligheid van totale verlamming met volle bewussyn te voorkom, word die pasiënt onder ligte narkose gehou. Hierdie oorspronklike metode is deur Lassen en sy medewerkers ontwikkel nadat hulle groot sukses behaal het met gevalle van bulbêre poliomiëelitis en asemhalingsversaking gedurende die poliomiëelitis epidemie van 1952 in Kopenhagen. Vroeë tracheotomie en aanhoudende positiewe druk-asemhaling het talle lewens gered. In 1954 het hulle verslag gedoen oor 4 gevalle van kaakklam wat met welslae op hierdie manier behandel is.<sup>5</sup> Die nadele is ooglopend. Die pasiënt wat vir 'n week of langer bewusteloos is verg die onafgebroke toesig van 'n bekwaame narkotiseur en die noulettendste uur-vir-uur verpleging—iets wat nie altyd in die praktyk uitvoerbaar is nie, afgesien van die ander belangrike gevare van kunsmatig veroorsaakte bewusteloosheid. Lassen en sy medewerkers het self onlangs die aandag gevestig op die ernstige hematologiese komplikasies wat deur langdurige verdoving met stikstofsukksied veroorsaak kan word.<sup>6</sup>

Die ander benadering tot die beheer van stuiprekkings wat tans weer ondersoek word, is die gewone een van kalmering. Die ideaal waarna hierby gestrewe moet word is 'n middel wat stuiprekkings van die spiere sal beheer sonder om die bewussyn of asemhaling te belemmer. Soos die meeste ideale sal hierdie een van 'n middel wat aan albei vereistes voldoen seker onverwenslik bly, maar belangrike sukses is reeds behaal met die barbiturate, met mephenesin, en onlangs ook met chlorpromazine. Parentale of binnearese gebruik van die barbiturate is reeds jare lank standaard behandeling, maar die asemhaling en bewussynsgaad word onvermydelik verlaag, en selfs met antibiotiese beheer en strenge verpleging kom komplikasies van die asemhalingsstelsel nogal dikwels voor. Dit is bekend dat mephenesin en chlorpromazine, wat albei hulle invloed uitoefen deur die geleiding van prikkels deur die senuweetussenweefsel te belemmer, doeltreffend is teen spierkrampe, en laasgenoemde is reeds met goeie gevolg gebruik by die beheer van kaakklamstupe in 'n kind.<sup>7</sup> Moontlik sal kombinasies van hierdie middels, aan ons doel beantwoord, soos die suksesvolle gebruik van onderbroke binnearese thio-pentone en senuwee-spier-blokkeringsmiddels wat Forbes en Auld<sup>8</sup> gerapporteer het.

exposed to all incoming excitatory impulses, and even trivial stimuli are now transmitted into activity. Unless the efferent nervous chain is broken or the patient sedated into physiological normality, this abnormal activity rapidly leads to exhaustion and death. Two kinds of clinical remedies have been applied. The more radical approach has been to induce total paralysis of all voluntary musculature with a neuromuscular blocking agent such as curare and—in order to obviate the emotional horror of being totally paralysed yet fully conscious—to maintain the patient in a state of permanent light anaesthesia. This novel method was evolved by Lassen and his colleagues after their success with cases of bulbar poliomyelitis and respiratory distress in the 1952 poliomyelitis epidemic in Copenhagen. Many threatened lives were saved by early tracheotomy and continuous positive-pressure respiration. In 1954 they reported 4 cases of tetanus that had been treated successfully in this way.<sup>5</sup> The drawbacks are obvious. A patient who is unconscious for a week or more requires the constant surveillance of a skilled anaesthetist and the most stringent round-the-clock nursing—something that is not really practicable, quite apart from all the other not inconsiderable hazards of induced unconsciousness. Moreover, Lassen and his colleagues have themselves recently drawn attention to serious haematological complications attributable to prolonged nitrous-oxide anaesthesia.<sup>6</sup>

The alternative approach to the management of convulsions that is being re-explored is the more usual one, viz. sedation. Here the ideal to be aimed at is the drug that will abolish, or at any rate control, muscle spasm without impairing consciousness or respiration. Whilst this combination, like most ideals, is probably unattainable, substantial success has been encountered with the barbiturates, with mephenesin, and more recently with chlorpromazine. Parenteral or intravenous use of the barbiturates has been standard treatment for many years, but respiration and the level of consciousness are inevitably depressed and the incidence of respiration complications, even with antibiotic coverage and strict nursing, is high. Mephenesin and chlorpromazine, both of which act by depressing conduction of impulses through the internuncial neurones, are known to be effective against the muscular spasm of tetanus, and good use of the latter drug has been made in controlling tetanus convulsions in a child.<sup>7</sup> Perhaps the answer lies in combinations of these drugs, such as the successful use of continuous intravenous thiopentone and neuromuscular blocking agents, reported by Forbes and Auld.<sup>8</sup>

1. Van die Redaksie (1954): S. Afr. T. Geneesk., **28**, 584.
2. Bvskrif (1956): Lancet, **1**, 493.
3. Scheibel, I. (1955): Bull. Wld. Hlth. Org., **13**, 381.
4. Brooks, V. B., Curtis, D. R. en Eccles, J. C. (1955): Nature (Lond.), **175**, 120.
5. Lassen, H. C. A., Bjorneboe, M., Ibsen, B. en Neukirch, F. (1954): Lancet, **2**, 1040.
6. Lassen, H. C. A., Henriksen, E., Neukirch, F. en Kristensen, H. S. (1956): *Ibid.*, **1**, 527.
7. Kelly, R. E. en Laurence, D. R. (1956): *Ibid.*, **1**, 119.
8. Forbes, G. B. en Auld, M. (1955): Amer. J. Med., **18**, 947.

1. Editorial (1954): S. Afr. Med. J., **28**, 584.
2. Annotation (1956): Lancet, **1**, 493.
3. Scheibel, I. (1955): Bull. Wld. Hlth. Org., **13**, 381.
4. Brooks, V. B., Curtis, D. R. en Eccles, J. C. (1955): Nature (Lond.), **175**, 120.
5. Lassen, H. C. A., Bjorneboe, M., Ibsen, B. en Neukirch, F. (1954): Lancet, **2**, 1040.
6. Lassen, H. C. A., Henriksen, E., Neukirch, F. en Kristensen, H. S. (1956): *Ibid.*, **1**, 527.
7. Kelly, R. E. en Laurence, D. R. (1956): *Ibid.*, **1**, 119.
8. Forbes, G. B. en Auld, M. (1955): Amer. J. Med., **18**, 947.

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## ACTIVE IMMUNIZATION AGAINST VIRUS DISEASES IN MAN

## A LECTURE\*

M. VAN DEN ENDE

*Professor of Bacteriology, University of Cape Town*

It is a well-known fact that the immunity elicited by virus infections is often very solid. Thus second attacks of chicken-pox and mumps are rare. In the case of measles second attacks have been observed, but some of these may be based on incorrect diagnosis and, in the rare authentic cases, many depend on unusual immunity reactions of the host. There are, however, virus infections in which immunity induced by an attack is very poor. We think here particularly of influenza and the common cold. It is worth while pausing to try and analyse the reasons for these differences in the effectiveness of the immunity induced, and to think of the behaviour of viruses in their host cells, before going on to a consideration of active immunization.

During the last decade virology has become a science on its own. An intensive study of certain bacterial, plant, animal and human viruses have brought to light many phenomena fundamental to the main problem we are considering today.

There was a time when, on the basis of the most striking and obvious lesions induced, we regarded viruses as having specific affinities for particular types of cells. Thus we thought of smallpox as a disease in which the virus multiplies selectively in the skin; and of polio virus as entering *via* the nerves of the naso- and oropharynx, from there being transported to the motor cells of the central nervous system, which it damaged or destroyed, thereby eliciting characteristic symptoms. We now know that the polio virus probably multiplies first in the gastro-intestinal tract and enters the blood-stream, only to be taken up in RE or other cells; from there increasing amounts of virus are released into the blood-stream, producing a secondary viraemia, and only then, in a small percentage of the total, does the CNS become affected. The vast-majority of individuals acquire the infection at an early age without untoward manifestations and with a lasting immunity. Unfortunately modern hygiene has brought about a changing epidemiological pattern in that infection tends to be delayed beyond infancy and, with the higher age incidence, there is a somewhat larger percentage with recognizable involvement of the CNS.

In the case of smallpox the classical work of Fenner on the corresponding disease in mice (*Ectromelia*) has thrown a great deal of new light. In this disease the primary site of infection is probably in the respiratory tract, but the classical signs and symptoms do not appear until there has been widespread dissemination as a result of primary and secondary viraemic stages. Skin lesions are but the superficial signs of a disease widespread in all the organs.

In measles, too, the eruption follows dissemination of the virus, probably from a primary inhalation infection. Why the rash associated with a disease like measles should have such a characteristic distribution, time of occurrence, etc. is far from clear. It may be that a good deal depends on an allergic reaction, and that it is not the mere presence of the virus which evokes the exanthem.

In sharp contrast to the infections in which viraemic stages are essential in the pathogenesis of the disease, stands influenza. Here the essential infection is limited to the respiratory tract—depending probably on the local spread along the surface of an infection originating in the respiratory tract. No doubt flu virus enters the circulation from time to time during an infection, but the essential disease-manifestations are independent of a viraemic stage.

New facts which have emerged from studies of the relation between viruses and their host cells also have an important bearing on immunity mechanisms. In this virus-host relationship at the cellular level we have learnt a great deal from the bacterial and plant virologist. Although it is dangerous to argue by analogy from bacteria to man there is sufficient evidence to indicate that similar mechanisms may apply.

The action of a fully virulent virus on a fully susceptible cell leads to intracellular multiplication of virus with destruction of the cell and release of a fresh brood of virus. The manifestations of this cell destruction may be immediately obvious in the production of a characteristic disease-picture. It does not, however, need much imagination to realize that there can be a great deal of such virus multiplication and cell destruction without a complex multicellular host like man or animal showing any symptoms.

Even, however, at the cellular level there are differences in virus-host interaction. In bacteriophages it is well-known that virus infections occur which do not do the host any apparent harm. In the so-called lysogenic cultures the bacteriophage infection remains almost entirely latent, and only in the occasional cell does it go on to multiplication and destruction of the host cell. In the intracellular state the 'phage remains quite unaffected by virus-specific immune serum introduced into the environment of the bacteria. An analogous situation occurs in herpes in man. In the human being the primary infection in herpes almost invariably occurs in early childhood—usually as a stomatitis, sometimes as a skin infection complicating, say, eczema. The primary infection is overcome, but many individuals thus infected remain herpes carriers for life. From time to time the appropriate stimulus may bring on an attack of herpes. Thus infection remains latent inside cells of the skin or mucous membrane—in spite of, or perhaps because of, the invariable presence in the

\* Delivered at the South African Paediatric Congress, Cape Town, 1956.

extracellular fluids of herpes antibodies. The herpes sufferer is immune from reinfection but his immunity is of no value in protecting the cells already permanently infected by the virus.

An even more interesting example of a latent infection is lymphocytic choriomeningitis in mice. In mouse colonies infected with this virus there is very little evidence of the presence of the virus—except the colony's insusceptibility to experimental infection, and the fact that virus can be recovered from apparently normal members of the colony. It is suggested that the L.C.M. infection is transmitted by infected mothers to the offspring during early foetal life—so early that the virus becomes established in the tissues and is not recognized as foreign protein. Antibodies to the virus are therefore not encountered in the mice, in spite of the silent presence of the virus. These mice are resistant to reinfection, not because of an orthodox humoral and cellular immunity, but because the latent virus excludes new and potentially infective virus.

Interesting differences in virus-host relationships are encountered every day by the virologist who attempts the adaptation of viruses to new hosts. Only a few examples need be quoted. Amongst the polio viruses one type (Lansing or Type 2) can be adapted to mice by intracerebral passages. In other words the intracerebral injection of the virus will cause paralysis and death and at post-mortem virus is found to have multiplied in the brain. The same virus injected intraperitoneally into the mice will, except very occasionally, elicit no signs of illness at all, although it almost certainly multiplies in the tissues of the host. Type-1 virus on the other hand, even when injected intracerebrally into mice, does not lead to any manifestations of disease. It can, however, be shown that even in these mice there has been a significant but transitory multiplication of virus.

In some cases virus may on administration to an animal produce manifestations of disease, but no new living virus can be propagated from the lesions by further passage. The virus has apparently succeeded in entering the cells, causing their destruction, but has not completed the cycle of reproduction. In other cases careful adjustment of experimental conditions has resulted in the release of virus which is detectable by *in vitro* methods but is incomplete in the sense that it is not infectious for a new host.

#### MECHANISM OF IMMUNITY

The attenuation of viruses for the original hosts by adaptation to new hosts probably depends on selection of mutants. It is a method which has been greatly exploited in veterinary medicine for the development of vaccines. It has at present, for reasons which will become obvious, relatively more limited application in human medicine. Blue tongue is a severe epidemic disease in sheep associated with a high mortality. The repeated passage of the virus in eggs increases its pathogenicity for the egg but at the same time reduces it for sheep—so that ultimately large doses of egg virus can be injected into sheep without eliciting symptoms. Attenuated strains have similarly been prepared of

horse-sickness, distemper, Newcastle disease and other viruses. In veterinary medicine it is easy enough to test whether such laboratory adapted strains are in fact attenuated for the original host. In human medicine it is not so simple. In poliomyelitis, for instance, we know now that of every hundred or more individuals infected only one will develop the classical paralysis of the disease. Even a fully virulent virus when fed to a hundred individuals may produce no paralytic infection. How many people would have to be exposed to infection before a strain of virus under investigation could be declared harmless? Almost inevitably, indirect methods have to be found to determine whether polio viruses are avirulent for man. Criteria on which the estimation of the harmlessness of strains can be based have been suggested by Koprowski and others. They depend on the response in monkeys and mice receiving inocula by various routes.

The attenuated strains multiply in the hosts into which they are injected but elicit no disease, whilst at the same time antibody production is stimulated. It is probably their multiplication which makes them such effective immunizing antigens. It has often been suggested that immunity in virus infection depends at least in part on the establishment of a permanent latent infection, but no direct evidence verifying this hypothesis has been forthcoming. Quite recently it has been shown by Ackermann that latent infections of cells in tissue culture by polio virus can be brought about by the addition of immune serum to the tissue-culture fluid. A similar mechanism may operate after recovery from natural infections in man and animals, or after the administration of living attenuated strains. It is of course a well-established fact that 'infection' of cells even by partially inactivated virus will exclude subsequently-added fully-virulent virus. The full significance and the extent of the operation of this interference phenomenon in naturally-acquired virus infections is unknown.

Evidence is accumulating also of the production by infected cells of serologically identifiable products other than the virus itself. Whether some or all of these are virus constituents produced in excess or simply by-products of virus synthesis remains to be determined. Their significance in bringing about active immunity is likewise still unknown.

The concept of a permanent latent infection of cells on which immunity may depend is at present almost entirely hypothetical. We should keep in mind the possibility that the latent 'virus' may be in a form not recognizable by the methods which have hitherto been applied, and which like prophage of lysogenic bacteria is intimately associated with cell constituents. Until such 'latent infections' in immune individuals are proven we shall be wise to disregard them in any consideration of practical methods of immunization, but I am bold enough to suggest that we shall hear more about them in the future.

Although specific antibodies detectable by neutralization and *in vitro* tests can be demonstrated in the serum after recovery from practically all virus diseases, there is strong evidence that those diseases which are associated with well-marked viraemia (chicken-pox, smallpox,

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mumps, measles) are followed by an effective and lasting immunity. On the other hand, influenza is followed by a poor immunity—probably to be accounted for by 2 important facts: (a) The virus spreads along the surface of the respiratory mucosa, where under normal circumstances, antibody concentration is less than in the blood-stream and effective contact with previously-formed antibody does not occur; and (b) the virus undergoes frequent significant changes in antigenic structure, so that antibodies against previous infecting viruses are ineffective against new strains.

#### ARTIFICIAL IMMUNIZATION

We can, I think, lay down certain principles concerning artificial active immunization. Thus we can reasonably anticipate that active immunization will be most effective in those virus diseases in which a viraemic stage is important in the pathogenesis, and in which only a single antigenic type of the virus is known. Furthermore, living attenuated virus is a better immunizing agent than killed virus. A striking example is the effective immunity induced against yellow fever by a single injection of living attenuated virus.

The viruses of blue tongue in sheep and horse-sickness occur in a multiplicity of serological types, so that break-downs of immunity induced with attenuated viruses usually depend on infection with a serological type not included in the multivalent vaccines employed against the diseases.

We could also suggest as a principle the opposite extreme, that active immunity will be induced with greatest difficulty against those diseases in which viraemia is not essential in their pathogenesis, and of which the casual viruses occur in an almost unlimited range of serological types; the striking example here is influenza.

Human medicine will of necessity have to continue to rely on killed vaccines for active immunization against a number of virus diseases which at present constitute unsolved public-health problems. The limiting factors in producing killed vaccines which evoke a significant antibody response are:

1. The ability to produce virus experimentally in high enough concentration. To be an effective antigenic stimulus a certain minimum bulk of antigen has to be administered. It may be that the amount required is significantly reduced by the use of adjuvants of the Freund type, but that the production problem remains significant is well exemplified by the experiences with polio vaccine.

2. The ability to purify the virus or to render it free of harmful antigens.

3. The ability to render the virus non-infective without destroying its antigenicity.

In 2 important virus diseases we are at an interesting stage of the development of prophylactic vaccines—poliomyelitis and rabies. South Africa is particularly concerned, not only because the diseases occur here, but because significant contributions to our knowledge of them have come from South African virologists working at Onderstepoort and the Poliomyelitis Research Foundation.

#### Poliomyelitis

In *poliomyelitis* it is not the infection of the gastrointestinal tract but the involvement of the CNS that leads to the disturbing manifestations of the disease. The CNS involvement is almost certainly preceded by viraemia. The polio virus occurs in any one of only 3 distinct serological types. Protection of the CNS by active immunization should therefore be relatively simple and effective. The development of an effective vaccine against poliomyelitis has therefore been a theoretical possibility for some years. All that was necessary was the production of the 3 viruses in sufficient concentration, free of harmful contaminating proteins (e.g. brain antigens), which on treatment with appropriate physical or chemical methods were rendered non-infectious; or the attenuation of strains in such a way that they became avirulent for man. The former type of vaccine has now been achieved—by the use of monkey kidney tissue cultures, and inactivation by critical concentration of formalin. Not only did the tissue-culture techniques make available a method for the ready cultivation of the polio viruses in large amounts, but the tissue culture virus is free of brain antigens with which virus cultivated in monkey CNS is inevitably contaminated.

The killed-virus vaccine can only be expected to reduce the incidence of paralytic polio—it cannot eradicate poliomyelitis from the community, and gastro-intestinal infections can be expected to continue much as before.

The practical difficulties which face the manufacturer of polio vaccine today are concerned with tests to ensure safety as well as potency, the complete elimination of kidney protein for fear of renal damage or Rh sensitization, and the avoidance of substances like penicillin to which abnormal sensitivity may be induced. The difficulties which face the manufacturers are large, but that they can be overcome as proved by the widespread use of the Salk-type vaccine in many countries today. The unfortunate accidents with this vaccine which occurred in the USA cast some doubts on the safety and efficacy. Further research can still be done to improve the potency and safety of the vaccine, but there can be no doubt that the results of trials undertaken in America in 1954 have been very encouraging and that the South African vaccination programme should be supported.

Several eminent virologists have expressed the view that the poliomyelitis problem will not be adequately solved until live attenuated strains have been developed—which can be fed to young children without risk of producing polio. The difficulty of deciding when a strain is sufficiently attenuated for human use has already been referred to. Furthermore the attenuated vaccine to be effective must establish an intestinal infection, but must not be capable of invading the CNS. Virus in the stools of vaccinated individuals would be infective for contacts—and there is a small risk that man-to-man passage might re-establish its original pathogenicity. In this respect poliomyelitis differs significantly from yellow fever and other arthropod-borne diseases. In these there is no phase during which

virus can be readily transmitted from the vaccinated individual to contacts. When the difficulties mentioned have been overcome vaccination against poliomyelitis may become simple and harmless. Children will then receive the 3 types of virus in attenuated form, by mouth, during early infancy when they still possess a passive immunity transferred from their mothers.

The administration of polio-virus vaccine by the oral route would have the additional advantage that it would follow the natural route of infection, and might thereby stimulate a local immunity. Such local immunity may perhaps be of greater and more general significance than is at present realized. With influenza the importance of local immunity of the respiratory epithelium, at least in experimental animals, has been shown by Fazekas de St. Groth. In mice, vaccination against flu by repeated intraperitoneal injection gives relatively slight protection against inhalation infection. Protection, however, is increased if the mice are given inhalations of homologous or heterologous vaccine after the intraperitoneal immunization. Such 'adjuvant' inhalation results in concentration of antibody in the respiratory epithelium.

#### *Rabies*

With rabies the problem is somewhat different. It is a disease which follows the introduction of virus into the tissues—usually by the bite of a rabid animal. Once the disease is established it is invariably fatal. There is, however, a latent period between the time of exposure and involvement of the CNS. During the latent period, the length of which depends on the severity and site of the lacerations, immunization can be undertaken. In South Africa we have for years employed a crude phenolized suspension of the brains of rabbits infected intracerebrally with a 'fixed' strain of virus. The source of most of our human rabies has until recently been wild carnivores in the northern Orange Free State and Western Transvaal. Recently however canine rabies has become a serious problem in the Northern Transvaal. An egg-adapted strain of rabies virus attenuated for animals has been developed, and this is being used effectively in the prophylaxis against rabies in dogs, but human vaccination is still carried out with the relatively unsatisfactory phenolized

rabbit brain. The potency of this vaccine has been questioned and its use is attended by the risk of neuro-paralytic accidents attributable to auto-immunization against brain antigens which it contains. Limited trials of the egg-adapted virus in human beings have been undertaken without untoward result. In my opinion there is little reason why the living attenuated virus should not shortly be in general use also for human prophylaxis. Unlike the case of polio the establishment of avirulence for man is relatively simple. Vaccination will not be widely administered because its use will be limited to individuals exposed; vaccinated individuals will not become infectious, so that man-to-man passage with reversion to virulence is unlikely. Furthermore there is as yet no evidence that cultivation of the virus in sufficiently high concentration for the preparation of killed vaccines can be achieved in tissues free from brain antigens.

#### *Other Diseases*

A great deal of work has been done in recent years on the development of vaccines against a variety of rickettsial and virus diseases. Much of this work is as fundamental as that which has been done on poliomyelitis but it has not had the same 'popular appeal'. The mumps virus for instance can now be cultivated in eggs almost as readily as the viruses causing influenza. The egg-adapted virus is attenuated for monkeys and man. Trials of the efficiency of the attenuated virus in eliciting active immunity against the disease have already been undertaken. An effective mumps vaccine would have a large potential public-health value.

The antibiotics have almost eliminated the dangers of the bacterial and rickettsial diseases. Antibiotics effective against virus infections have not yet been isolated, and for some years to come we shall remain dependent on the virologists and immunologists for protection against virus diseases.

Perhaps the time is not far off when our infants will receive not only a subcutaneous injection against diphtheria and a scratch against smallpox, but also a mouth spray against mumps and a variety of respiratory viruses, as well as a cocktail against poliomyelitis and the flock of intestinal viruses now being isolated.

## READING DISABILITY IN CHILDREN\*

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Dyslexia, or reading disability, is a syndrome which is characterized by an inability to read properly even though the individual may have normal or superior intelligence.<sup>1</sup> By definition any hearing, visual or emotional defect is excluded as a cause of this condition. However, the very fact of such a disability leads to widespread emotional upset. 'Like a particle dropped into a super-

saturated solution even a small disability can act as a nucleus round which other things crystallize' (*Lancet*).<sup>2</sup>

The inability to read, then, is no minor handicap. It has far-reaching emotional and mental effects, which are all the more unfortunate since the condition is remediable.

That dyslexia is not an uncommon condition is evidenced by the fact that the incidence is between 10% and 15% in American school children.<sup>3</sup> Many

\* A paper presented at the South African Paediatric Congress, Cape Town, 1956.

doctors, psychiatrists and educationalists have attributed this to the 'look and say' method of teaching reading. It is stated that in countries like Germany where the old phonic method is still used, this problem is a very minor one. While I feel there may be some truth in this statement, it is far from the whole truth; I believe there is a specific reading disability or syndrome. It may be true that more children are recognized with this condition because of the 'look and say' method, whereas formerly the minor degrees would catch up, or simply take a little longer to learn to read. Logically, it seems that the older (phonic) method is more sensible—after all, the child first learns about words and their meanings by hearing them from his mother's lips. Mankind itself got along without reading for a couple of million years and the use of the spoken word is an altogether older accomplishment than the use of the written word; so why not carry on logically from there with our teaching methods. I should like to repeat, however, that the method of teaching is only a contributing factor to reading difficulties.

This problem is far wider than is generally believed, and in this country many children are floundering with reading and emotional difficulties, the true nature of which is not recognized. Surely, if the incidence is 10% to 15% in American children, a similar percentage must be expected in our children; yet I have not come across a single article in South African medical literature, and have very rarely heard the subject discussed by teachers, psychologists or pediatricians, and certainly never by medical students.

Case finding usually depends on the teachers or parents but, as these children are often described as lazy or retarded, the true diagnosis is often completely missed. It behoves us, then, as doctors, to be the case finders—indeed with a knowledge and awareness of the syndrome one can make the diagnosis in the pre-school period.

There are many examples in the literature of intelligent children and even adults who could not read, or had learnt to read with the greatest difficulty. I have seen 6 cases in the past year, all mild; they had all been taught by the 'look and say' method, and I feel they probably would not have come to the doctor had they been taught by the old phonic method. Their progress would have been slower than that of the average child, but not so slow as to cause any alarm. The more severe cases, however, make no progress by any of the ordinary methods of teaching. John Hunter, according to Bakwin,<sup>3</sup> did not learn to read until he was 17 years old.

#### SYMPTOMS

The reading disability is specific. The acquisition of reading skill lags behind the other scholastic achievements. It does not come up to the expectations ordinarily justified by the child's mental age. Terms used for it in the literature are strephosymbolia (twisted symbols), dyslexia, and congenital word-blindness. Orton<sup>1</sup> objected to the last term since, he says, the condition is not congenital and there is no blindness.

These children before they enter school are usually considered very bright, and Park<sup>4</sup> vividly describes 2 children, John and Mary, who were veritable pre-

school geniuses. I cannot do better than quote his lucid description:

'Before Johnny entered school he was the talk of the neighbourhood, and a little wonder. At Sunday-school on festive days he recited long poems with nary a bobble. His intellect seemingly was without bounds. The multiplication table was 'duck soup'. Sure, he could rattle off the alphabet.

In short, Johnny had photographic ears. What he learned at his mother's knee was his for ever. It was the same way with the near-prodigy Mary. Before she saw the inside of a schoolroom, Mary played the piano at recitals; of course it was by ear—but she performed with delight. It appeared nothing was too difficult for Mary's auditory faculties to circumvent—and retain.

Folks said Johnny would be a Justice of the Supreme Court or a United States Senator. Mary indubitably was headed for top flight leadership in a world of women. How could they miss?

Chapter 2 finds Johnny and Mary starting to school. Kindergarten was a lot of fun. Then came First Grade, where the child is taught to distinguish words from certain combinations of letters—reading. Brace yourself! Johnny and Mary flunked.

Children of average mentality and below average IQs in their classes picked up reading soon enough. There were just no two ways about it, Johnny and Mary could not bridge the gap between the printed primer and their previous superior performance. Patient teachers couldn't impart the simple faculty to their nimble little brains; special tutors fell down. What in the world was the matter?

The detective student will say, "Why, that's simple. The kids need glasses. How in the world can you teach that C A T means cat when their eyes are completely out of focus because their vision is distorted?"

Shock Number 2. Johnny and Mary had their eyes examined. Expert oculists agreed that each tested out 20-20, which is perfect vision by medical standards.'

In their pre-school years these children enjoy being read to and show normal interest in letters and numbers. During the first couple of years at school they do not learn to read, but very often, as they are bright, they give the impression of being able to read because they make good use of their memory. The condition then may not be recognized in the kindergarten classes, and in the so-called progressive schools it may pass unrecognized for as long as 4 years.

Letters, syllables and whole words are often reversed. More commonly individual letters are written backward and sometimes upside down. In rare instances, the child may have mirror reading, i.e., can only read from right to left. He can copy well and will often make up the text from the pictures in the book.

Later on, subjects like history and geography are also affected, because the child can't use his text-book, or if he does manage to read, struggles so that he has no time to actually learn the work. While he may be good at arithmetic in the early classes, even this becomes impossible later on because he can't read.

Abnormalities in lateral dominance are common. Very many of these children are ambidextrous, but usually in a clumsy fashion, and mixed dominance such as right-handedness with left-footedness and left-eyedness are common.

Speech disturbances, too, are common and occur in about 50% of cases. Talking is often delayed, and stuttering, lisps, impure and cluttered speech, are frequent findings.

The condition often appears in families whose members show a high incidence of alteration in lateral dominance, speech disturbances and reading difficulty.

Emotional difficulties, as can be expected, very often form the most important part of this syndrome, and so

is very often the reason why the child is taken to the doctor. Downes and Schuman<sup>5</sup> report some of the ways in which these emotional difficulties may manifest themselves. The most obvious thing about the child is that he hates being made to read, and may panic or stammer if he is given a reading test. Other distress signals, they note, are 'knee jerking, nail biting, paper tearing, face fussing, and hair pulling'. Again, he may use bad language, cheat, steal, destroy property, or bully. At home he does anything but read, preferring the radio, television, bird watching, telephoning his friends, or even helping with the washing up. He plays with younger children and prefers the company of the 'maid, the handyman and the not-so-nice children down the road'. His sense of being different, of having a gap in his faculties, may be very alarming to him. 'There seems to be a deep-seated, pervasive, terrifying kind of disunity in the world of a disabled reader.' He tends to be emotionally immature and knows it; and his school mates know it.

He may become solitary or enter with real gusto in the things he feels he can do well, such as athletics, drawing and mechanics. On the other hand, feeling inadequate and lacking expert help in overcoming his difficulty, he may become deeply discouraged. He comes to equate good reading, school success and high marks with parental approval and love. This anxiety for approval may be transferred in later life to his employer or to society. He may become a perfectionist, unwilling to risk a mistake, and his dread of making one may limit more and more the field in which he dare trust himself. Such a child makes no trouble for parent or teacher. 'He finally never makes any trouble, or anything else for that matter.'

The severity of this abnormality varies greatly. Some children are only mildly affected and they seem to improve rapidly. In others the abnormality is severe and may persist well into adolescence or even adult life. Boys are affected 4 times as often as girls. The age when the condition is first recognized depends on the alertness of the teacher and the concern of the parent; and on the doctor, especially if the child is brought to him for a specific complaint such as an emotional one. If the doctor finds in the history a story of speech difficulty, alteration in lateral dominance, and instances of other members of the family having had difficulties of language function, then he must be on the look-out for this syndrome. Special tests are available for young children, to test out reading ability, e.g. in recalling a row of pictures—the order in which they are memorized is important.

#### ETIOLOGY

Hinshelwood,<sup>6</sup> in 1917, believed that specific reading-disability is caused by injury or faulty development of the left angular gyrus in right-handed persons. He stated that any condition diminishing the number of cortical cells within this area or interfering with the blood supply would lower the functional activity of the centre and hence would diminish the power of retention of the visual images of words and letters, which is an absolutely essential accomplishment of the act of reading.

Reading, speaking and writing is controlled, initiated or overseen largely, if not entirely, from one hemisphere of the brain. Any defect in the dominant half of the brain, however produced, is likely to result in extensive damage to the language function, while injury to the corresponding area in the other half of the brain generally gives no specific language-symptoms whatever. The brain hemisphere which has major control over the language function is also the hemisphere which controls handedness, eyedness and footedness. Although there are exceptions, it is the rule that in right-handed people the left hemisphere of the brain is the dominant one and *vice versa*.

Persons with a specific reading-defect have trouble or difficulty only in the complex function of word recognition. They are able to see and interpret objects correctly. Thus children with this condition can learn single letters. The difficulty is not of vision, but of comprehension.

In some instances the inability to read appears to be a manifestation of a developmental lag, similar to a delayed development in speech. These children may not begin to read until they are 10-15 years old, when they seem suddenly to acquire this ability without any special instruction. Some observers believe that the emotional disturbances antedate the reading difficulties, and then when parents and teachers bring pressure on these children they become anxious and insecure and react in the manner discussed above. This idea, however, cannot explain the great preponderance of boys over girls, the typical reversals, and the contrast between the disability to read words and the ability to read numbers and musical notes.

In the differential diagnosis one must exclude visual, hearing and mental defects. Children with severe visual defect, particularly with errors of refraction, may present with reading difficulties. Poor hearing, which leads to the hearing of blurred or indistinct sounds, produces a very similar picture. Cases of these kinds, of course, do not present the full syndrome.

#### MANAGEMENT AND TREATMENT

Early recognition of this condition is imperative because, as I have tried to point out, there may be far-reaching mental disturbances. Therapy is both educational and psychiatric, but one naturally must correct any co-existing visual or hearing defect. The parents should be taught to understand the nature of the disability, and when they are told they are often so relieved that their child is not mentally affected that this in itself helps the child enormously. One has to gain the child's confidence and restore its self-confidence. He should be assured that his intelligence is normal and that his inability to read is no evidence of stupidity. His success in other fields should be emphasized and applauded and any special ability encouraged. The teacher should be experienced and enthusiastic. One has to combine all three methods of teaching—visual, auditory and kinaesthetic. The method of kinaesthetic or motor discrimination is often neglected; here the pupil traces words, copies letters in their proper sequence, feels block letters, and feels the position of the

tongue and the lips while pronouncing words. The pupil's errors should be analysed and the teacher can then lay stress on the type of instruction which will help the individual pupil most. During this period the child's confidence is to be bolstered up at all times.

#### CONCLUSION

In this paper I have tried to show that reading disability is a specific defect and is part of a syndrome, which includes not only difficulty in reading, but also speech difficulties, alteration in lateral dominance and often a family history of language disorders. I have also emphasized that the emotional effects may be the presenting and often the most pressing problem of these cases.

In conclusion, I would like to quote once more from the *Lancet*:<sup>2</sup> 'It is unfortunate that such a mild and common deviation from the usual pattern should be

allowed to become the centre for a host of disabilities. A better understanding of the causes of backwardness in reading, and more skilled management of those who display it, might save much unnecessary trouble both for teachers and taught. It seems worth trying to discover these children early and give them the kind of help they need.'

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## HAEMOLYTIC DISEASE OF THE NEWBORN

### A REPORT FROM THE NATAL RHESUS UNIT

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In 1951, the Director of the Durban Blood Transfusion Service, Dr. J. C. Thomas, put forward the suggestion that a 'Rhesus Unit' should be established at Addington Hospital, Durban, to serve not only Durban but the whole Province of Natal, and to which all Rhesus-immunized women in the province might be referred for their confinements. The advantages of such a scheme seemed obvious in that adequate facilities, both laboratory and clinical, are much more readily available at a large specially-equipped centre than in outlying country districts or even in private nursing homes. The concept of centralization of these difficult cases was wholeheartedly endorsed by the obstetrical and paediatric staffs of the hospital and, accordingly, an explanatory circular was sent to all medical practitioners in the province inviting their cooperation. In the Durban area the response to this appeal has been excellent and it is probably a true statement that in the past 4 years the vast majority of Rhesus-immunized women in the area have been delivered at Addington Hospital. The use made of the unit by the country districts is more difficult to assess, since the number of cases diagnosed in these districts is problematical. Despite the relatively small number of country cases, however, it is encouraging to note that these have come from all parts of the province, and the majority have done well.

At Addington Hospital there is a close liaison between the obstetrical and paediatric departments, and all

infants born there come under the care of the paediatric staff, who are thus responsible for deciding on and carrying out the treatment of the newborn infant with haemolytic disease.

The procedure adopted by the Rhesus unit has been standardized as far as possible. The prospective mother's blood group is determined during early pregnancy. If she is Rh-negative antibody tests are carried out and repeated at 24, 32 and 36 weeks if practicable. Should Rhesus antibodies develop admission to hospital is advised in the 38th week.

Immediately after delivery clotted and unclotted samples of cord blood are sent to the special Rhesus laboratory, where the following tests are carried out:

1. The direct Coombs anti-human-globulin test
2. Haemoglobin estimation
3. Blood and Rhesus grouping
4. Serum bilirubin estimation
5. Normoblast count.

Whilst the above tests are being carried out in the laboratory the newborn infant is subjected to a careful clinical examination, during which particular attention is paid to the weight, the state of the general health, the presence or absence of jaundice, and the size of the liver and spleen. Details of the mother's obstetrical history are also carefully assessed. Essential laboratory and clinical investigations are usually complete within an

hour of birth and it is possible, therefore, to institute the required treatment with the minimum of delay.

#### *Town and Country Cases*

The series under consideration comprises a total of 112 cases admitted to Addington Hospital during the 4 years 1951-55. Of these, 84 were admitted from the Durban area, the remaining 28 being referred from various country towns throughout the province of Natal.

#### *Stillbirths etc.*

The total of 112 cases includes 12 pregnancies which terminated in hydrops foetalis, stillbirth or abortion. For the purpose of this paper these regrettable cases will be excluded from our analysis, which is concerned primarily with the management of the affected live-born infant. Suffice it to observe that, where there is a history of one or more stillbirths and where the husband is homozygous, early induction of labour or Caesarean section might possibly increase the chances of procuring a live infant. Such a policy was not generally adopted in this series but will be carefully considered in future cases, bearing in mind the apparent advantages of early induction in selected cases as reported by Kelsall and Vos.<sup>1</sup>

#### *Live-born Infants*

During the 4 years 100 live-born infants were born of Rh-sensitized mothers. Although antibodies were detected in the maternal blood, 20 of these infants proved to be Rh-negative and, being unaffected, did not require any treatment. The remaining 80 Rh-positive infants were all affected to a greater or less degree and their treatment will be discussed in some detail with particular reference to the indications for exchange transfusion.

The total number of cases presenting in the 4 years may be tabulated as follows:

Stillbirths and abortions	..	12 cases
Infant Rh. —, Coombs test —	..	20 cases
Infant Rh. +, Coombs test +	..	80 cases
Total	..	112 cases

#### *Indications for Exchange Transfusion*

In the treatment of haemolytic disease of the newborn the merits of exchange transfusion have been proved by a number of investigators.<sup>2,4</sup> Compared with other forms of treatment not only is the survival rate higher, irrespective of the birth weight or of the severity of the disease,<sup>4</sup> but the incidence of kernicterus is reduced by at least two-thirds. Impressed by these undoubted virtues, some units still perform exchange transfusion in all cases where the cord blood gives a positive Coombs anti-human-globulin test. Not only is this a waste of precious Rh-negative blood, but it exposes unnecessarily a considerable proportion of infants (estimated at 30-40% of those affected) to the risks of the operation, viz. shock, air-embolism, infection, incompatible transfusion, portal-vein thrombosis and perforation. Apart from the first we have as yet encountered none of these mishaps, but they are too real to be ignored.

In an attempt to define the indications for exchange

transfusion Mollison and Cutbush<sup>5,6</sup> first drew attention to the value of the cord haemoglobin as an index of severity, taking 14.8 g.% as the critical value. Subsequent studies<sup>3,4</sup> on their untreated infants revealed an incidence of kernicterus of 7% in the mature and 14% in the immature groups, and these workers have, therefore, revised their criteria and now regard as indications for immediate exchange transfusion (in the presence of a positive Coombs test):<sup>7</sup>

1. A birth weight of 6 lb. or less, or an infant born 3 weeks or more before the expected date of delivery (irrespective of other findings)

2. A history of the mother having previously given birth to an affected infant (irrespective of other findings)

3. A cord haemoglobin below 15.5 g. %

They point out that in cases where the cord haemoglobin lies between 15.5 and 17.5 g. % there is still a possibility of kernicterus occurring, and in such cases the onset of jaundice within the first 24 hours should be an indication for treatment. Walker and Neligan,<sup>8</sup> discussing this same problem, regard the cord bilirubin level as being of considerable help, and in cases where the cord haemoglobin lies between 14.8 and 17.7 g. % a bilirubin value of 2.8 mg. % or above is taken as an indication for exchange transfusion. Other factors which are recognised as being of some assistance in assessing border-line cases are: The degree of positivity of the Coombs test, the maternal anti-Rh titre and the nucleated red-cell count on the cord blood. We would also include splenic and hepatic enlargement.

In the Durban unit we have not favoured the practice of routine exchange transfusion in all affected infants but have attempted to differentiate between those requiring immediate treatment and those which could safely be left untreated. Sometimes the selection of cases for treatment presents little difficulty, as in the severely affected infant whose precarious state is apparent from the moment of birth. More often the decision entails the careful appraisal of a variety of factors. Whilst following fairly closely the indications for exchange transfusion suggested by other units, our standards may have been less rigid in that the decision for or against treatment has not always depended on pre-elected laboratory findings. For example, although the cord haemoglobin level has proved a most valuable guide in all cases, we have not necessarily been bound by any arbitrary figure but rather have considered the haemoglobin level in relation to the many other relevant factors. A further point is that the ultimate decision regarding treatment in each case has rested on one individual, a policy which, we believe, makes for uniformity and consistency of approach.

It is now proposed to discuss the foregoing guiding principles in the treatment of haemolytic disease of the newborn in relation to this series of cases. To simplify description the latter have been divided into 2 main groups, viz.: *Group I*—immediate exchange transfusion performed (50 cases). *Group II*—no immediate treatment given (30 cases).

*Birth Weight.* There were 14 infants whose weight was less than 6 lb. at birth. Of these, 12 were given immediate exchange transfusion and 2 were left untreated. One of the treated cases with a birth weight of only



Fig.  
detec

4 lb. 14 oz. died, and the remainder made a good recovery. We do not feel convinced, provided the other factors are satisfactory, that all infants weighing 6 lb. or less at birth require immediate treatment.

**Mother's Obstetrical History.** As already mentioned, the previous obstetrical history of the mother must influence the decision whether her infant should receive

careful assessment of other factors. It is of interest to note that one case which was left untreated and died of kernicterus had a cord haemoglobin of 16.9 g. %.

**Serum Bilirubin.** Fig. 2 illustrates the serum-bilirubin values of the cord blood which was estimated in the

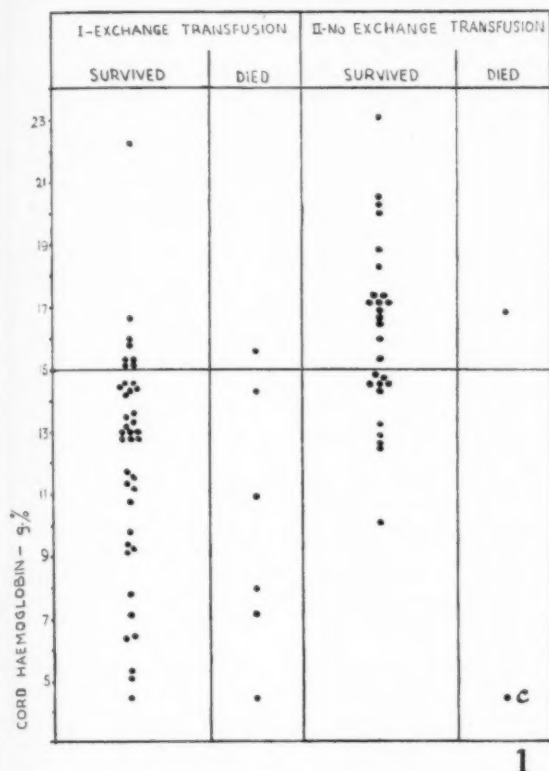


Fig. 1. Cord haemoglobin levels. C=due to anti-c, undetected antenatally.

immediate treatment. In this series 16 mothers gave a history of having had one previous infant affected and in 4 of these the new infants were left untreated and made an uneventful recovery. A further 11 mothers had had more than one affected infant and in all these cases immediate exchange transfusion was carried out. It is suggested that a history of only one previous infant affected is not an absolute indication for immediate treatment, but that where there have been 2 or more infants affected exchange transfusion should usually be carried out.

**Cord Haemoglobin.** Fig. 1 shows that the majority of cases having exchange transfusion had cord haemoglobin levels below 15 g. %. Those above this level had other indications for treatment. It will be noted that 11 cases with haemoglobin levels below 15 g. % were left untreated and made a good recovery, and it is probable that a considerably higher proportion of cases in this category could be spared unnecessary operation by

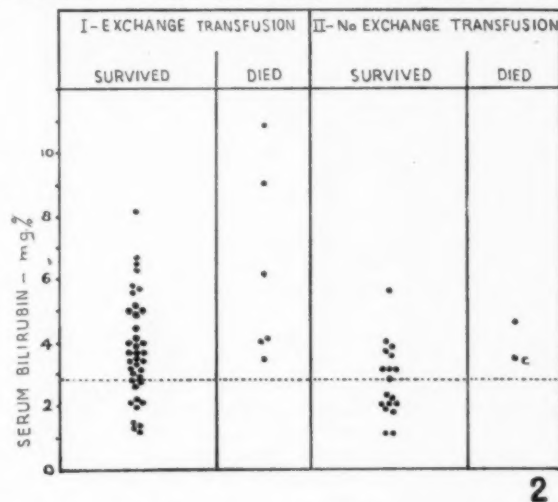


Fig. 2. Serum bilirubin levels. C=due to anti-c, undetected antenatally.

majority of cases. Though all the fatal cases had values above 3 mg. %, it will be noted that over 50% of the non-fatal cases also had values above this figure. A serum bilirubin of 4 mg. % or over would, however, appear to have some prognostic importance, since 28% of our cases in this category proved fatal.

The association between very high serum-bilirubin levels and kernicterus is well-established,<sup>9</sup> and it would therefore appear advisable to perform exchange transfusion and, if necessary, to repeat this procedure whenever the serum bilirubin rises to 20 mg. %, and perhaps less in premature infants. We have had occasion to repeat the exchange transfusion in one instance, with a successful outcome.

**Degree of Positivity of the Coombs Test.** This has been estimated in 4 degrees (in nearly all cases by the same technician), and the distribution in the 2 groups is shown in the following table:

Coombs Test	Group I		Group II	
	Successful	Fatal	Successful	Fatal
++++	26	5	9	1
+++	8	1	4	—
++	3	—	5	1
+	7	—	10	—

The association of a strongly positive Coombs test with the need for exchange transfusion, and particularly with the fatal cases, is evident. No further conclusions can be drawn from this small series, but it is felt that in doubtful cases a strongly positive Coombs test should swing the balance in favour of exchange transfusion.

**The Maternal Antibody Titre.** Weiner<sup>10, 11</sup> has for long fought rather a lone battle in defence of the maternal

anti-Rh titre as an index of the severity of the disease in the infant, although Mollison<sup>7</sup> has recently observed that a titre of over 64 can be correlated with an increased risk of kernicterus. Our own figures lend considerable support to Weiner's view as will be seen from the table.

Maximum Maternal Anti-Rh Titre (Albumin)	Group I		Group II	
	Successful	Fatal	Successful	Fatal
8,192	2	1	1	—
4,096	6	—	2	—
2,048	8	1	2	1
1,024	4	1	2	—
512	5	1	2	—
256	6	1	4	—
128	8	1	2	—
<hr/>				
64	1	—	7	1
32	1	—	1	—
16	1	—	4	—
8	1	—	—	—
4	1	—	—	—
2	—	—	—	—

It will be noted that 45 of the 50 cases requiring exchange transfusion had a maternal antibody titre of over 64, and all the fatal cases, with one exception, belonged to this group. In view of these findings we feel that in doubtful cases a maximum maternal antibody titre of more than 64 is a definite pointer in favour of exchange transfusion.

**The Normoblast Count.** This we have found to be a very variable and somewhat unreliable test, but a count of 10 or more per 100 white cells has usually been associated with severe disease. Our results show that, of the cases requiring exchange transfusion, 93% had a normoblast count of 10 or more, whereas in those cases which were left untreated only 62% had a count in this range. From a practical point of view, however, we consider the normoblast count to be of little value.

**Splenomegaly.** The degree of splenomegaly, where it was accurately recorded, may be depicted as follows:

Splenomegaly	Group I		Group II	
	Successful	Fatal	Successful	Fatal
++	8	4	—	—
+	19	2	5	—
0	12	—	20	1

It will be noted that 33 of the cases in group I, or 73%, had splenomegaly, as compared with only 20% in group II. Moreover, in the latter group there were no cases which exhibited gross splenic enlargement. We are well aware of the difficulty in assessing splenic enlargement in the newborn, but experience leads us to believe that a readily palpable spleen is usually indicative of severe disease.

To summarize this section concerning the various factors which influence the decision whether or not immediate treatment of the affected infant is indicated, it is suggested that there is a considerable group of cases with cord haemoglobin levels below 15 g.%, in which fine judgement can lead to a decrease in the number of exchange transfusions performed. In such cases knowledge of the birth weight and of the mother's

obstetric history is of prime importance, and valuable guidance is obtained from the serum-bilirubin level, the maternal antibody titre, the positivity of the Coombs test, and the size of the spleen.

#### TECHNIQUE OF EXCHANGE TRANSFUSION

In the majority of our cases exchange transfusion was carried out by way of the umbilical vein in accordance with the technique originally advocated by Diamond in 1947.<sup>12</sup> It must be recorded, however, that in 5 cases where exchange transfusion was considered advisable it was found impossible to establish a satisfactory flow from the umbilical vein. In 2 of these a polythene catheter of 0.5 mm. bore was passed into an umbilical artery and a successful exchange performed by this route. We suggest that this technique is well worth attempting when difficulty is encountered with the umbilical vein. Approximately 70-80 c.c. per lb. body-weight of group-O Rh-negative blood were given and the same amount withdrawn. Immediately before the operation the donor blood was concentrated by decanting about 15% of the plasma. Latterly, after every 100 c.c. exchanged, 1 c.c. of 10% calcium gluconate has been injected into the vein.<sup>13</sup> Warming of the blood was not a routine precaution but it is probably advisable. Marting *et al.*<sup>14</sup> quote Wheeler's interesting suggestion that the transfusion of cold blood may precipitate cardiac arrhythmias. Although in this series no obvious cardiac irregularities developed, 2 infants showed signs of collapse towards the end of the transfusion but quickly revived when warmed up in an incubator. In fact, one of the difficulties has been to maintain body heat during a somewhat lengthy procedure, and the giving of warmed blood might help to resolve this difficulty.

A repeat exchange transfusion was given on the second day in one case when the serum bilirubin rose to 21 mg.% and jaundice became intense. Since the umbilical vein was no longer patent the exchange was successfully carried out through the saphenous vein in the right thigh,<sup>15</sup> and the infant made an excellent recovery.

#### RESULTS

The results obtained in this series of 80 infants affected with haemolytic disease of the newborn may be described briefly as follows:

**Group I.** In this group, comprising 50 cases, immediate exchange transfusion was performed. Complete recovery occurred in 43 cases, or 86%. One further case survived but is suffering from the effects of kernicterus, and the remaining 6 cases died. Four of the cases in this group required subsequent direct transfusions.

**Group II.** In this group of 30 cases no immediate treatment was given, although 7 were given subsequent direct transfusions during the neonatal period, when the haemoglobin showed signs of falling rapidly. This was a precautionary measure and possibly was not necessary in all cases. In this group, 28 infants recovered completely and 2 died.

The total results are shown in the following table:

Group	Cases	Deaths	Mortality %	Kernicterus (surviving)
I. (exchange transfusion)	50	6	12.0	1
II. (no exchange)	30	2	6.6	—
Totals	80	8	10.0	1

The over-all mortality of 10% is reasonably satisfactory and compares favourably with results from many other centres. The mortality figures include one case where death may not have resulted from haemolytic disease since autopsy revealed gross congenital defect of the left kidney and ureter. Another fatal case was due to anti-c and was undetected before death (see below). These 2 somewhat doubtful cases have been included in the over-all mortality rate of 10%.

*Short summaries of the fatal cases* are shown at the end of this article; these reveal some interesting facts which may serve as a guide to reducing mortality in future cases.

For those babies who died after exchange transfusion perhaps little could have been done, but in the light of more recent knowledge they might have been given the benefit of a second or third exchange if and when the serum bilirubin rose to 20 mg.%,<sup>16, 17</sup> even though signs of kernicterus had already appeared. Moreover, it is just possible that those deaths that occurred within 24 hours of transfusion (cases D and F) might have been associated with electrolytic disturbances,<sup>18</sup> though the blood used was not old. The injection of calcium gluconate now forms part of our routine.

There remain the two cases (G and H) in which the patients died without exchange transfusion. Case G was the first affected child, was not premature, and had a cord haemoglobin of 16.9 g.%. Jaundice was first noticed 36 hours after birth. On Mollison's criteria, therefore, there was no indication for exchange transfusion, but Walker would have been influenced in its favour by the serum bilirubin of 4.4 mg.%. In retrospect we consider that the very strongly positive Coombs test and the high maternal antibody titre of 2,048 were additional factors which should have influenced us in favour of performing exchange transfusion in this case.

Case H was due to anti-c and occurred in an Rh-positive (D) woman who had received a blood transfusion 6 years previously. We feel that death in this instance was inevitable, since the incompatibility was unsuspected and the infant died 20 minutes after birth. Nevertheless, this unfortunate case must surely underline the danger of indiscriminate blood-transfusion in female children and young adults, and illustrates the necessity of searching for unusual antibodies in Rh-positive women who give a history of previous blood-transfusion.

#### KERNICTERUS

It will be noted in the summaries of the 8 fatal cases that signs of kernicterus were evident before death or at autopsy in 4. The majority of the surviving cases have been followed up for at least 6 months and, so far as it has been possible to ascertain, only one of these exhibits signs attributable to kernicterus. This infant was quite severely affected at birth with a cord haemoglobin of 12.0 g.% and serum bilirubin of 5.8 mg.%,

although there was no obvious jaundice. The Coombs test was strongly positive. Immediate exchange transfusion was performed and the infant was discharged from hospital apparently vigorous and well. Subsequently convulsive attacks became frequent and there is now gross mental defect and blindness. A happy sequel for the mother of this tragic case has been the recent birth of a healthy unaffected infant.

#### FOLLOW-UP

We are fortunate in being able to follow-up these cases in the clinic at Addington Hospital for babies born in the maternity section. Weekly haemoglobin estimations are carried out in all cases of haemolytic disease, and it is thus possible to observe their progress during the first few months of life. The majority of infants in this series have been followed up for at least 6 months.

The usual haemoglobin pattern has been a fairly steady fall until the 6th or 7th week, followed by a spontaneous and sustained rise. Provided the infant is thriving satisfactorily, our practice has been to withhold blood transfusion unless the haemoglobin level falls below 6 g.% (40% Haldane).

#### SUMMARIES OF FATAL CASES

##### Case A

First child normal; second, third and fourth children died of congenital haemolytic disease; fifth child unaffected.

*Sixth child.* Caesarean section. B.W. 10 lb. Cord Hb. 14.2 g.%. Cord bilirubin 4.0 mg.%. Coombs +++++. Hepato-splenomegaly ++. E.T. 825/800 c.c. Well, but slight jaundice on the following day. Signs of kernicterus on 3rd day. Increasing jaundice and death on 4th day. P.M. refused.

##### Case B

First child normal. Second child jaundiced and died on 3rd day.

*Third child.* B.W. 7 lb. 1 oz. Cord Hb. 4.4 g.%. Cord bilirubin 4.4 mg.%. Coombs +++++. Slight jaundice with mottling of skin. Hepato-splenomegaly +. E.T. 850/800 c.c. Condition poor at end of exchange transfusion, but improved. On 3rd day respiratory difficulty, and oliguria and haematuria and oedema of legs developed; still jaundiced. On 4th day skin petechiae, haemoptysis and death. P.M.—Pulmonary and bladder haemorrhage; kernicterus.

##### Case C

*Second child.* B.W. 8 lb. Cord Hb. 15.7 g.%. Serum bilirubin 3.6 mg.%. Coombs +++++. Splenomegaly ++. E.T. 700/700 c.c. Increasing jaundice from 2nd day. Kernicterus and death on 4th day. P.M.—Kernicterus and pulmonary haemorrhage.

##### Case D

First 2 children normal. Third child given exchange transfusion.

*Fourth child.* B.W. 7 lb. Cord Hb. 7.2 g.%. Cord bilirubin 6.2 mg.%. Coombs +++++. Jaundiced with ecchymoses on face and back. General condition poor. E.T. 700/700 c.c. Deteriorated during transfusion. Died 18 hours after birth. P.M.—Hepato-splenomegaly; scattered pulmonary haemorrhages; no kernicterus.

##### Case E

First two children normal. Third and fourth children stillborn.

*Fifth child.* B.W. 7 lb. 10½ oz. Capillary Hb. 10.9 g.%. Cord bilirubin 10.8 mg.%. Coombs +++++. Deeply jaundiced. Splenomegaly ++. General condition poor. E.T. 730/730 c.c. Deterioration during procedure, with temporary improvement later. Died 20 hours after birth. P.M.—Cerebral haemorrhage right ventricle; left hydronephrosis with grossly dilated ureter.

##### Case F

First, third and fourth children alive. Three stillbirths and 2 miscarriages.

*Seventh child.* Elective Caesarean section at 39 weeks. B.W. 4 lb. 14 oz. Cord Hb. 8.0 g.%. Cord bilirubin 9.1 mg.%. Coombs +++++. Jaundiced. Hepato-splenomegaly ++. E.T. 425/405. Condition improved after transfusion. Grunting respira-

tion with blood-stained mucus oozing from mouth 7 hours afterwards. Fine crepitations heard at left lung base. Died 12 hours after transfusion. P.M. refused.

#### Case G

Maternal antibody titre—2,048 (incomplete).  
*Second child.* B.W. 6 lb. 11½ oz. Cord Hb. 16.9 g.%. Cord bilirubin 4.4 mg.%. Coombs +++++. No jaundice or hepatosplenomegaly. Exchange transfusion not done. Slight jaundice first noticed 36 hours after birth, which then increased. Signs of kernicterus on 4th day. Died on 6th day. P.M.—kernicterus and pulmonary haemorrhage.

#### Case H

Maternal blood-transfusion 6 years previously.

*Second child.* Antibodies not detected antenatally. B.W. 7 lb. 12½ oz. Cord Hb. 4.4 g.%. Cord bilirubin 3.5 mg.%. Coombs +++. Antibodies type anti-c detected—titre 64 (incomplete). Died 20 minutes after birth. P.M.—intracranial haemorrhage; liver and spleen +; scattered haemorrhages, especially retroperitoneal; jaundice; no kernicterus.

#### SUMMARY

The procedure adopted at the Natal Rhesus Unit, centred at Addington Hospital, Durban, is described from its inception in 1951 to 1955.

The number of cases of haemolytic disease of the newborn encountered during this 4-year period is shown, and the indications for exchange transfusion are discussed.

Results obtained in infants subjected to immediate treatment and in those left untreated are shown, the over-all mortality for the series being 10%.

The incidence of kernicterus is described.

Reference is made to the progress of affected infants

after leaving the hospital, the majority having been followed up for at least 6 months.

Short summaries of the fatal cases are included.

We wish to record our thanks to Dr. J. V. Tanchel, Medical Superintendent of Addington Hospital, for permission to publish this report; to Mr. Harold Renton, Senior Visiting Obstetrician to the hospital, for maintaining faith in our judgement of these cases; and to Dr. J. C. Thomas who, as Director of the Durban Blood Transfusion Service, organized the Rhesus Laboratory Service.

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## MYASTHENIA GRAVIS IN TWO BANTU CHILDREN

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Myasthenia gravis is rare in the Bantu. One case has been reported in an adult.<sup>1</sup> The present cases are believed to be the first reported in Bantu children.

#### CASE I

On 5 January 1955 a 5-year-old female Bantu child was admitted to the Baragwanath Non-European Hospital, for investigation of bilateral external ocular paresis. The mother said the child had been well until 1 month before admission, when she noticed that the child's eyelids were drooping and that the eyes were not moving freely in all directions. On direct questioning she said the eyes appeared bigger in the early morning; also that the child could play tirelessly all day. She had not noticed any dysphagia or slurring of speech. There had been no preceding trauma or illness. There was no similarly affected person in the family.

*Examination.* The child was well nourished and showed marked bilateral ptosis and some sagging of the lower jaw. All the external muscles of the eyes showed weakness, but the external recti were affected to the most. No exophthalmos was present. The pupils were equal and reacted to light and accommodation. The fundi were normal. There was no evidence of hyperthyroidism. Muscle fatigability could not be demonstrated clinically, owing to lack of cooperation from the small patient. As far as it was possible to assess, no diplopia was present. The tendon jerks were normal and no muscle wasting could be detected. The rest of the physical examination was negative. A test dose of 0.5 mg. of Prostigmine was given by intramuscular injection and within 15 minutes ptosis was abolished and the range of eye movements was considerably improved (Fig. 1).



Fig. 1. (a) Case 1, before Prostigmine. (b) Case 1, 30 minutes after Prostigmine.

The urine was normal, the blood count normal, and the blood Wassermann negative. The serum potassium was 19 mg. per 100 c.c.

On X-ray of the chest the heart and lungs appeared normal, and no mediastinal mass was seen.

*Treatment.* Mestinon, 80 mg. by the mouth augmented by

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A 3-year 1955 w left side active a noted. toward

Fig. 2 after

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1/8th gr. of ephedrine was given 6-hourly. The improvement which was noted within 30 minutes lasted for 3-4 hours. The child was maintained on this dosage until discharge and no side or untoward effects were noticed. At no time during the stay in hospital, was weakness noted in any other part of the body. The patient was discharged on 12 February 1955, with marked improvement of the external ocular paresis on the above dosage.

## CASE 2

A 3-year-old Bantu female child was admitted on 11 October 1955 with a history of drooping of the eyelids, particularly on the left side, for 3 weeks before admission. The child was physically active and no disturbance of articulation or mastication had been noted. The parent volunteered that the weakness was maximal towards the end of the day. Before this illness, the child had



Fig. 2. (a) Case 2, before Prostigmine. (b) Case 2, 30 minutes after Prostigmine.

enjoyed good health. There was no other member of the family with a similar complaint.

Examination confirmed the ptosis, which was more marked on the left. The eye movements were limited in all directions and there was marked sagging of the lower jaw. The rest of the physical examination was normal. A test dose of 0.5 mg. of Prostigmine, together with 1/150th gr. of atropine, was administered by intramuscular injection, with a dramatic result. Ptosis of the right eye was completely abolished and that of the left eye considerably improved. The child was able to move her eyes freely in all directions and the jaw no longer drooped. Similar investigations were carried out as in case 1 and yielded no abnormal results. Muscle reactions were tested. The contractions of the orbicularis oris muscle became sluggish after 90 contractions produced by rapid faradic stimulation, which suggested a mild myasthenic reaction.

**Treatment.** The patient was well controlled on 15 mg. of oral Prostigmine plus 1/8th gr. of ephedrine 6-hourly. In view of the poor prognosis, it was decided to submit the child for thymectomy. The operation was performed on 7 December 1955 and the post-operative course was uneventful. On the day following operation, the Prostigmine was reduced to 7½ mg. 6-hourly, but this dose proved inadequate and, 1 week later, the original dose was reverted to. The child has required this dosage ever since. Histological section of the thymus showed this organ to be within normal limits.

## DISCUSSION

In typical cases the characteristic symptoms are those of fatigue in voluntary muscles, increased by exertion. The muscles affected vary from case to case and, in a particular patient, from time to time.

The extrinsic muscles of the eyes are almost constantly

affected in varying degree and may remain exclusively involved for many years. The mask-like or drowsy facies, is mainly due to the drooping eyelids and relaxation of the facial musculature. As the condition progresses, there may be generalized weakness with marked restriction of activity. In children the symptomatology is similar to that of adults. The loss of strength is not so great as in the muscular dystrophies. The patients are never so weak as to 'climb up themselves'. Remissions may occur, varying from several weeks to years. Death may result from the inhalation of food, from respiratory failure, or as the result of intercurrent infection.

Kibrick,<sup>2</sup> reviewing myasthenia gravis in childhood, classified cases into 2 groups:

**Type 1—Transitory myasthenia of the newborn.** In this group, infants born to mothers suffering from myasthenia gravis may present shortly after birth with hypotonia, inability to suck and difficulty in coping with secretions. Administration of Prostigmine may be life-saving in these cases. These infants recover completely after neonatal survival.

**Type 2—True congenital myasthenia gravis.** In this group, the signs of the disease may be present in infancy, but the cases differ from those in type 1, in that the onset is not as severe or as generalized. The mothers of infants in this group do not themselves suffer from the disease.

## Differential Diagnosis

1. Post-diphtheritic paralysis was considered. There was no history of preceding illness. Paresis of the external ocular muscles, without any of the more common manifestations such as palatal palsy and weakness of the extremities and muscles of the neck, rendered this an unlikely diagnosis.

2. The after-effects of poliomyelitis was included in the differential diagnosis, but here again it was felt that the localization of weakness to the external ocular muscles excluded this possibility.

3. Familial periodic paralysis was considered. In this condition weakness commonly affects the proximal limb muscles and tends to occur after periods of rest. Tendon reflexes may disappear during episodes of paralysis. There is usually a familial incidence. The blood potassium is low. None of these features were present in these cases.

4. In meningo-vascular syphilis paresis is more widespread. Pupillary abnormalities, stigmata of congenital syphilis, mental deterioration, and a positive blood Wassermann, may be expected.

The typical history of increased weakness following exertion, the absence of wasting in affected muscles, with no evidence of sensory impairment, together with a dramatic response to Prostigmine, point conclusively to the diagnosis of myasthenia gravis.

I wish to thank Dr. E. Kahn and Dr. S. Wayburne, and Dr. J. D. Allen, Superintendent, Baragwanath Hospital, for permission to report these cases.

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# THE INCIDENCE OF CERTAIN STRAINS OF *E. COLI*, *SHIGELLA* AND *SALMONELLA* IN KWASHIORKOR IN THE PRETORIA AREA

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Although many cases of kwashiorkor present with diarrhoea, in a previous investigation<sup>1</sup> of 180 cases only 13 *Shigella* or *Salmonella* were isolated. Others<sup>2</sup> have also encountered gastro-intestinal upsets in a large proportion of cases and have been no more successful in isolating recognized pathogens.

It is by no means certain whether the diarrhoea in these cases must be considered a precipitating factor, or whether it is merely a common feature of the disease. Gopalan<sup>2</sup> has given suggestive proof of the causative role of infective diarrhoea by showing that in the South of India the peak incidence of kwashiorkor follows the fly and infective diarrhoea season regularly by a period of 1-2 months.

Because of our failure to isolate intestinal pathogens in more than a small fraction of cases, and in view of our finding 'pathogenic' *E. coli* in about 40% of cases of gastro-enteritis in this area during the summer months,<sup>3</sup> it was decided to investigate the incidence of these organisms in cases of kwashiorkor.

## MATERIAL AND METHODS

Rectal swabs were obtained from 106 cases of kwashiorkor and 69 control cases during the summer months of October 1955—April 1956. Apart from 19 cases which were admitted to the wards, the patients were seen in the Out-patient Department, where the swab were obtained. Most of the cases presented with a short history of illness and diarrhoea, and appeared to be mildly affected. The swabs from the 69 control cases were also obtained in the Out-patient Department, from non-kwashiorkor patients with no symptoms referable to the gastro-intestinal tract.

The age-incidence of both groups was between 6 months and 4 years. A second swab was obtained at an interval of 7 days from the 19 admission cases.

According to the history obtained from the mother more than half the kwashiorkor cases had diarrhoea for periods varying from days to months. The difference in the concept of normal bowel action between the Bantu and European makes this history completely unreliable.

The methods of obtaining rectal swabs, delivery to the laboratory, and subsequent bacteriological investigation were precisely the same as in the previous investigation.<sup>3</sup> Apart from the recognized intestinal pathogens (*Salmonella* and *Shigella*) the presence of *E. coli* strains 026B6, 055B5, 086B7, 0111B4, 119B? and 128B12 were specifically investigated.

## RESULTS

Table I represents the incidence of organisms isolated in the kwashiorkor and control groups and shows that

TABLE I—ORGANISMS ISOLATED

Organisms	Kwashiorkor Cases (106)	Control Cases (69)
<i>E. coli</i> :		
026B6 .. .. .	6	2
055B5 .. .. .	0	0
086B7 .. .. .	2	2
0111B4 .. .. .	3	3
0119B? .. .. .	0	4
0128B12 .. .. .	10	1
<i>Salmonella</i> :		
typhi-murium .. .. .	4	0
paratyphi C .. .. .		2
<i>Shigella</i> :		
flexneri		
Type 2 .. .. .	2	1
Type 3 .. .. .	2	0
boydi Type .. .. .	1	0
Total .. .. .	30	15
% Cases in which organisms isolated	28.3%	21.7%

there is no significant difference in the 2 groups as determined by the normal distribution test. The results of the repeat swabs are set out in Table II, and show

TABLE II—REPEAT SWABS OF ADMISSIONS

Case No.	Organisms present on admission	Organisms present after 7 days
1	086B7	Negative
2	Negative	"
3	026B6	"
4	Negative	"
5	0128B12	"
6	Negative	"
7	"	"
8	<i>Shigella boydi</i> 2	"
9	0128B12	"
10	<i>Shigella flexneri</i> 2	"
11	<i>S. typhi-murium</i>	"
12	Negative	<i>Shigella flexneri</i> 3
13	<i>S. typhi-murium</i>	Negative
14	Negative	"
15	0128B12	"
16	Negative	0119B?
17	"	Negative
18	"	"
19	"	"

that in the majority of cases the organisms initially present were not isolated on the second occasion. These patients were all on sulphadiazine and penicillin therapy for the entire interval period. Two possible ward infections are illustrated in cases 12 and 16.

## SUMMARY AND CONCLUSIONS

The majority of kwashiorkor cases have diarrhoea and it is tempting to think that the debilitating effect of a bout of diarrhoea has at least a precipitating effect on the disease. The patients usually presented with a short history of diarrhoea and the chances of detecting pathogens were thus possibly optimal.<sup>4</sup> Despite this, our results indicate no significant difference in the incidence of *E. coli* strains, *Salmonella* and *Shigella* organisms in cases of kwashiorkor and a control group of patients. The *E. coli* strains searched for in this investigation are by no means proved pathogens in the gastro-intestinal tract but, assuming that they are, an infective cause for the diarrhoea was found in only 28% of the cases of kwashiorkor.

From this limited investigation it thus appears unlikely that *E. coli* and other pathogens play an important causative role in the diarrhoea of kwashiorkor.

We wish to thank Professor Davel for allowing us access to the patients and Miss D. Hamman for technical assistance rendered.

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## KWASHIORKOR

A short report of the Second Congress of the South African Paediatric Association, held in Cape Town on 19-21 January 1956, was published in the *Journal*\* a few weeks ago. In the

present issue several papers presented at the Congress are published, and the following is a summary of the reports submitted in a discussion on Kwashiorkor:

## AMINO ACIDS AND KWASHIORKOR

J. D. L. HANSEN, M.B., ChB., M.R.C.P., D.C.H.

Cape Town

Dietary therapeutic trials with synthetic amino acids in kwashiorkor were described. Previous work had shown that vitamin-free casein was capable of curing kwashiorkor. It was now demonstrated that cure could be initiated with a mixture of 18 synthetic amino acids when combined with salts, vitamins and dextrose. A similar mixture, but containing only 11 amino acids, was also capable of curing the disease. If vitamins were removed from this latter mixture improvement was much slower and less com-

plete. It would seem that when amino acids are provided at marginal levels vitamins may permit their more efficient utilization.

Preliminary studies on the excretion of amino acid in the urine in cases of kwashiorkor have as yet failed to reveal any abnormality when compared with controls on the same diets.

The possibility that amino-acid imbalance or specific amino-acid deficiency might play a part in the pathogenesis of kwashiorkor was discussed.

## MEGALOBLASTOSIS IN KWASHIORKOR AND OTHER DISEASES

F. P. WALT, M.R.C.S., L.R.C.P., D.C.H.

Durban

At McCord Zulu Hospital, Durban, in association with Dr. S. Holman (late Clinical Pathologist, Durban Medical School), who examined all the bone-marrow smears, it was found that megaloblastosis occurred in over 5% of all admissions, and that more than half of the megaloblastosis cases were suffering from kwashiorkor:

Period	Total Admissions	Megaloblastosis	
		Megaloblastosis	with Kwashiorkor
17.2.54 to 7.2.55	779	42 (5.4%)	22 (52% of 42)
14.2.55 to 7.1.56	764	41 (5.5%)	31 (74% of 41)

Of 143 cases of kwashiorkor 22% showed megaloblastosis.

A monthly analysis of the 83 megaloblastic cases showed that the incidence was greater in the summer months of December 1954 and January 1955, as follows:

1954		1955		1954		1955	
January	..	..	9	July	..	2	4
February	..	2	5	August	..	3	2
March	..	4	6	September	..	1	5
April	..	4	2	October	..	2	1
May	..	0	6	November	..	3	0
June	..	4	5	December	..	7	6

\* Report (1956): S. Afr. Med. J., 36, 436.

A controlled series was dealt with from 14 February to 5 June 1955 (excluding 23 days over Easter vacation), in which bone-marrow aspirations were performed on 201 consecutive admissions, with the following results:

	Control Cases	Megaloblastic Cases
Number	.. 32	19 (9.5% of 201)
With Kwashiorkor	.. 9 (28%)	16 (84% of 19)
Total Deaths	.. 4 (12.5%)	6 (31.6% of 19)
Kwashiorkor Deaths	.. 1 (3.1%)	4 (21% of 19)
Reticulocyte Peak	.. 6 (19%)	19 (100%)

By January 1956, the 575 bone-marrow aspirations that had been performed revealed an incidence of 7.1% of megaloblastosis.

Megaloblastosis was diagnosed by bone-marrow examination, a haemoglobin almost always below 6.7 g.%, and a clinical picture showing severe anaemia on admission, or anaemia developing insidiously or suddenly in 'crisis'.

The cases were treated with folic acid, 5 mg. *tds* orally or by intramuscular injection, which caused the bone marrow to return to normal within 72 hours. Some cases also needed blood transfusions.

The conclusion drawn from this study is that all cases of kwashiorkor should be given folic acid for at least 14 days from the commencement of treatment.

## KWASHIORKOR IN CAPE TOWN

PETER V. SUCKLING, M.D., M.R.C.P., D.C.H.

*Cape Town*

1. Despite a return to their poor home surroundings, which encourage the disease, children with kwashiorkor in Cape Town usually make a good recovery.

2. Quick recovery takes place from oedema, hypo-albuminaemia, changes in the skin and mucous membrane, pancreatic dysfunction, electrocardiographic changes, and disorders of temperament; and there is no evidence of permanent damage.

3. Growth in height is maintained at the same rate as in controls, and though at a lower level does not differ statistically from those controls. Growth in weight shows that there is recovery in 2

years from the malnutrition found at admission, and that thereafter it progresses at the same rate as in controls.

4. Anaemia is corrected more slowly, but recovery is complete in 5 years.

5. Liver biopsies show that the liver can apparently recover completely from the fatty change of kwashiorkor. The histological features of cirrhosis were consistently absent, though one child had suggestive clinical signs. Other microscopical abnormalities in the biopsy sections were infrequent and slight.

## KWASHIORKOR : A STUDY TO DETERMINE THE IMPORTANCE OF VARIOUS POSSIBLE ETIOLOGICAL FACTORS

J. G. A. DAVEL, M.B., B.Ch., F.R.C.S.Ed., M.R.C.P.Ed., D.C.H.

*Pretoria*

The vast majority of cases fall between 6 months and 3 years, and the boys and girls are about equal in number. It occurs much more in the Bantu than in the Coloured or Indian population.

The diet is almost exclusively cereal—as mealie meal—very little meat, fish, eggs, milk or cheese being consumed. The social

Dr. E. JANSSEN, *Cape Town*, also took part in the discussion and described his experience of various feeding regimes in kwashiorkor. He discussed the general management of these difficult

background is that of poverty, overcrowding, ignorance and prejudice.

Many of the cases were cared for by relatives, mostly grandparents, while the mothers worked.

Infection plays an important role either as a precipitating incident, or because of lowered resistance, and is mainly of the respiratory, alimentary or urinary systems.

and described his experience of various feeding regimes in kwashiorkor cases and stressed the value of protein milk and acidified milks.

## SOCIETY FOR INDUSTRIAL HEALTH, CAPE TOWN SUB-GROUP

A meeting of the sub-group was held on 20 June, when addresses were given by Dr. James Marshall and Dr. J. G. Louw.

## INDUSTRIAL DISEASES OF THE SKIN

Dr. James Marshall spoke on this subject. He defined these conditions as 'all those modifications of the skin seen in workers which result from, or are aggravated by, direct contact with irritants—solid, liquid or gaseous, and of animal, vegetable or mineral origin'.

'It is often assumed', he said, 'that the majority of industrial skin diseases are allergic reactions; this is not so, and only about 20% are due to allergy'.

The study of industrial skin diseases is no new subject and such conditions were recognized centuries ago by Paracelsus, Ramazzini and Patissier; but it is only in the last 50 years that they have assumed major importance as a result of increased use of synthetic rather than natural products. Skin diseases now represent probably the most important group of industrial accidents, with between 50% and 80% of all reported cases. In the 1920s about 800 cases of industrial dermatoses were reported annually in Britain; the numbers have now risen to about 20,000.

Primary irritants account for most cases of industrial diseases of the skin and can produce a great variety of lesions. Petrol and oil products, alkalis and cement account for 20-40% of such cases. It is important to remember that materials used for cleansing the hands after work are often more liable to cause trouble than things handled in actual work.

It is impossible to deal with the countless causes of allergic contact-dermatitis, which usually manifested itself as an exzema. The common causes varied from one country to another and mention was made of sensitivity to certain woods common in South Africa. Allergic reactions to substances applied in the treatment of industrial accidents are common and Dr. Marshall deplored the use of sulphonamide, penicillin and anti-histamine ointments. The important subject of patch testing in cases of allergic dermatitis was discussed; all substances used by the worker should be so tested and not only the major suspects.

The various methods of protecting the skin against irritants were described and the point was made that these measures are more important for the healthy than for the already affected worker. Once a worker has dermatitis, especially of the allergic type, protective creams etc. are of little value. Workers may become sensitive even to barrier creams.

Selection of personnel for hazardous industries, the treatment of established dermatoses and the fate of workers with industrial skin diseases were briefly discussed.

## THE EYE IN INDUSTRIAL PRACTICE

Dr. J. G. Louw spoke on this subject. He stated that eye injuries were the commonest condition with which industrial doctors were called upon to deal. Not only must they diagnose and treat them but they should advise employers and workers on first aid and prevention. Ninety per cent of eye injuries were preventable.

Selection of personnel in industry is important. A minimum visual acuity of 6/12 is safe for indoor work. Workers showing 6/24-6/36 should only be employed on outdoor jobs. A one-eyed man should not be engaged in mining, hammering, drilling or chipping.

Protective goggles should always be worn where abrasive operations are carried out, and where radiation and light are dangerous. Workers sometimes dislike wearing goggles because when they become scratched they obscure vision. The newest type of safety goggle has several layers of transparent plastic material, each of which can be removed as it becomes damaged, thus ensuring good vision.

All tools used should be of the best quality and in good repair. The common eye accidents were listed as follows:

Corneal foreign body (by far the commonest type of injury)  
Corneal and conjunctival abrasions and burns (by hot metal and fluids)

Perforating injuries

Chemical burns

Contusions, which may involve the retina, lens or optic nerve.

Conjunctivitis or keratitis.

Special industrial poisons such as lead, methyl alcohol, chloroethylene.

Damage by radiant energy.

Miners' nystagmus.

In treating a foreign body it is important to remove the rust ring which forms rapidly round the foreign body and causes more irritation than the foreign body itself. In Intra-ocular foreign bodies the degree of injury will depend on the chemical composition of the foreign body and its size, weight and rate of velocity. The great majority embed themselves in the cornea or sclera. In diagnosis, history is most important; doctors are sometimes misled by the patient's own interpretation of the accident, but an intra-ocular foreign body should be suspected if there is the slightest hint of the possibility. Examples were given of unsuspected foreign bodies revealed by X-ray.

First-aid treatment of chemical or irritant injury or burns is of the utmost importance. Immediate, copious and prolonged irrigation with tap water is required. There is no time to get antitoxins. Eye fountains, faucets for self-irrigation, or cupped

hands, must be used. For the treatment of lime particles embedded in the eye, removal with a cotton-wool applicator, and irrigation for at least an hour with 5% ammonium tartrate or chloride, is needed, as these particles cause delayed damage.

Although figures for eye injuries reported as industrial accidents are not available in South Africa, the cost is known to be high. Some interesting data had been collected by a sixth-year medical student of the University of Queensland, Australia. His research revealed that the indirect cost of all industrial accidents is 4 times higher than the cost of compensation and medical fees.

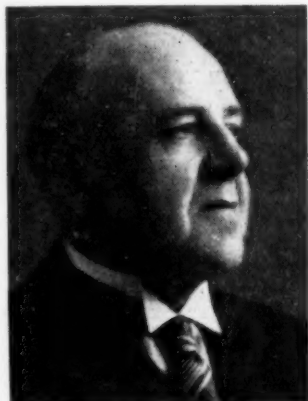
Workers most likely to sustain eye injuries are skilled workmen. Compensation paid is a fraction of the regular salary. A man risks a 33 1/3% cut in salary for the rest of his life each time he sustains an eye injury. The indirect cost to the community in supporting blind and semi-blind persons is considerable. Added to this is the personal suffering and mental distress of the injured worker.

Industrial doctors could do a great deal toward prevention of injury by suggestions regarding lighting and colour, selection of personnel, and visual surveys such as those that are being carried out on a wide scale in industry in America.

## IN MEMORIAM

JOHAN COENRAAD GIE, M.B., B.S. (LOND.), M.R.C.S., L.R.C.P.

Dr. J. C. Gie, who died at Sea Point, Cape, on 15 June 1956, was born at Griquatown, Griqualand West, in 1892. He was educated at the South African College School, Cape Town, and at the University of London (Guy's Hospital) where he took the conjoint qualification in 1917 and his London degree in 1918. He held house appointments at Guy's Hospital and elsewhere in England, and served as Temporary Surgeon, R.N., in the first World War.



Johan Coenraad Gie

In 1919 Dr. Gie returned to South Africa. After working for a time in the Pathology Department of the University of Cape Town he began practising in Sea Point in 1922, where he continued until his last illness. For some years he was Honorary Medical Officer at the Somerset Hospital.

He leaves 2 sons, one a doctor and one an engineer, and a married daughter living in England.

*Dr. A. W. S. Sichel of Cape Town, writes:* The many friends of Jack Gie, and particularly those who served with him on the Council of the Cape Western Branch and on Federal Council, heard of his passing with deep regret, and lament his loss.

In acceding to the request to write an appreciation of my old friend and colleague I will confine my remarks to the work he did for the Association, leaving it to others to refer to his professional career, his war service, and his love for and pride in his old medical school, Guy's Hospital.

My friendship with Gie began in May 1934, when he first became a member of the Council of the Cape Western Branch, on which he served continuously until his forced retirement in 1951, due to ill-health. Perusal of the Branch Council minutes reveals how assiduously he took part in its activities and how rarely he absented himself from meetings of the Council and the numerous committees of which he was a member.

His interest in contract practice no doubt was awakened when he was elected to the Workmen's Compensation Committee of the Branch in 1938. His competence in handling the special problems which devolved on the Committee was recognized by his election as Chairman in 1940.

In 1942 he was chosen to be one of 4 members of a special Subcommittee set up by the Branch Council to deal with the question of Medical Aid Societies, in which field the United Banks Medical Aid Society was a pioneer. At that time the Medical Aid idea was in its infancy. From the side of the Medical Association it can be said with truth that Gie was the father of Medical Aid. Those of us who worked in close contact with him in those early days realize full well all that he did to establish and promote the Medical Aid Society scheme as we know it today. To him almost alone, and in large measure because of his personality, is due the achievement of bringing about the Association's recognition of Medical Aid Societies which were willing to enter into an agreement acceptable to both parties. As the movement grew so did Gie's enthusiasm and the extent of his labours; and he did not spare himself.

On his election as a member of Federal Council in 1945 it followed as a matter of course that he was appointed a representative of the Cape Western Branch on the first Central Committee for Contract Practice to be established in October 1946. It is not surprising that he was chosen as the Convener of this important Committee, a post which he held until his retirement in 1951.

During these 5 years the greater part of the work of the Central Committee for Contract Practice was conducted from the Head Office, and it devolved on Gie to do what amounted almost to a full-time job in an honorary capacity while at the same time carrying on a large and very busy general practice. There is no doubt that the onerous nature of this work, and the conscientious way in which Gie applied himself to it, played no small part in precipitating the stroke which laid him low.

His loyal service was not unattended by honour, for he became President of the Cape Western Branch in 1940 and in recognition of his valuable work for the Association he received the well-merited award of the Association's Bronze Medal in 1950.

In the course of my long experience as a worker for the Association I can say with truth that I was never associated with a more loyal and cooperative colleague. To know Jack Gie was to love him. His whole personality exuded geniality and enthusiasm, attributes which explain his popularity both with his medical colleagues and with officials of the Medical Aid Societies with whom he came in close contact.

Gie's life was not without tragedy and the culminating blow came when his wife and helpmate, who had nursed him through his long illness with loving care, passed away only a short while before the call came to him. It may be well that he went so soon to re-join her. His fortitude and cheerfulness during his last illness, known only to his intimates, will be an inspiration to all who have to face adversity.

We salute the memory of Jack Gie in the knowledge that he has

received and deserved that final commendation, 'Well done, thou good and faithful servant'.

Dr. C. A. H. Green, of Johannesburg, writes: To have known the late Dr. J. C. Gie was both a privilege and an inspiration. His unfailing kindness, courtesy and unselfishness stood out like beacons. Service for others came naturally to him, without any thought of reward or glory for himself. Instead of taking the opportunity to relax he devoted all his spare time to affairs connected with the Medical Association. Only those who worked with him knew how much of his time this work took up, and how much this took out of him. There can be no doubt that this shortened his life by many years. He himself, however, would have been the last person to regret this. Dr. Gie was one of those who never failed to see the other person's point of view. If it be true that 'Manners maketh man' then he unquestionably was a man. The world in general and the medical profession could ill afford to lose such a lovable man.

Mr. K. C. W. Lambert, General Secretary, United Banks' Medical Aid Society, and Chairman, Southern Council of Medical Aid Societies, writes: The passing on of Johan Coenraad Gie severs one of the few remaining golden strands which link the great Medical Aid Society system of today with its modest and unspectacular beginnings.

This kindly man, whose tact and wisdom were so much in evidence in the early days when 'medical aid' was in its infancy, who used to be at the beck and call of all young Medical Aid Societies in their growing-pains, could perhaps not have foreseen just where his care and guidance were going to lead them and others who came later. For, 30 years ago, his home was closely identified with the development of the one great society which I have the honour to serve and on which so many others have based their own organization.

And so it is perhaps not too much to say that had it not been for Dr. J. C. Gie's influence a wrong road might have been taken, a road where the relationship between the doctor and patient might so easily not have been reproduced as it is in today's unseen but very real cooperation between the Medical Association of South Africa and the Medical Aid Societies.

Those of us who were privileged to attend the first joint meetings between the Association and the Societies will ever remember his kindly understanding and sympathy with us in our problems and difficulties. The hours he devoted to them, without thought of reward save that of knowing that a job was being well done, were long and many.

As a wise master builder he laid the foundations. 'Si monumentum requiris circumspecte'.

## OFFICIAL ANNOUNCEMENT : AMPTELIKE AANKONDIGING

### APPOINTMENT OF EDITOR

Applications are invited for the post of Editor of the *South African Medical Journal*. Applicants must be registered medical practitioners having knowledge and experience of medical journalism. A knowledge of languages will be a recommendation. The salary attaching to the post is on the scale £1,800×60—2,400, plus cost of living allowance of £352 for married men and £176 16s. 0d. for unmarried persons. (£100 of this allowance will be consolidated for pension purposes). The commencing notch will be according to experience, at the discretion of the Federal Council.

In addition to the Association's official *Journal*, the successful applicant will be required to edit the quarterly '*South African Journal of Laboratory and Clinical Medicine*'. He will also be required to join the Association's Superannuation Fund.

Applications, together with testimonials and a certificate of health, should be addressed to the undersigned to reach him before 31 August 1956.

A. H. Tonkin  
Secretary

Medical House  
35 Wale Street  
Cape Town  
19 May 1956

### APPOINTMENT OF ASSISTANT SECRETARY

Applications are invited from bilingual, registered medical practitioners for the post of Assistant Secretary of the Medical Association of South Africa. Although the successful applicant may be required to work in the Transvaal, it is likely that he will be expected to spend some time initially at the Association's Head Office in Cape Town.

The salary attaching to the post is on the scale £1,250×50—1,750, plus cost of living allowance of £352 for married men and £176 16s. 0d. for unmarried persons. (£100 of this allowance will be consolidated for pension purposes). The commencing notch will be according to experience and will be determined by the Federal Council. The successful applicant will be required to join the Association's Superannuation Fund.

Applications, together with testimonials and a certificate of health, must reach the undersigned on or before 31 July 1956.

A. H. Tonkin  
Secretary

Medical House  
35 Wale Street  
Cape Town  
19 May 1956

### AANSTELLING VAN REDAKTEUR

Aansoeke word ingewag vir die betrekking van Redakteur van die *Suid-Afrikaanse Tydskrif vir Geneeskunde*. Applikante moet geregistreerde geneeshere wees met kennis en ondervinding van die geneeskundige joernalistiek. 'n Kennis van tale sal 'n aanbeveling wees. Die salaris aan die pos verbonde is op die skaal £1,800×60—2,400, plus 'n duurtetoelag van £352 vir getroude mans en £176 16s. 0d. vir ongetroude persone. (£100 van hierdie toelag sal vir pensioendoelindes by die salaris gekonsolideer word.) Die beginsalaris sal na goeddunke van die Federale Raad met inagneming van vorige ondervinding vasgestel word.

Die applikant sal ver wag word om benewens die redaksie van die Vereniging se amptelike *Tydskrif* ook dié van die kwartaalblad, '*Suid-Afrikaanse Geneeskundige Tydskrif vir Laboratorium- en Kliniekwerk*' op hom te neem. Hy sal ook by die Vereniging se pensioenfonds moet aansluit.

Aansoeke, vergesel van getuigskrifte en 'n gesondheidsertifikaat, moet aan die ondergetekende gerig word om hom vóór 31 Augustus 1956 te bereik.

Mediese Huis  
Waalstraat 35  
Kaapstad  
19 Mei 1956

A. H. Tonkin  
Sekretaris

### AANSTELLING VAN ASSISTENT-SEKRETARIS

Aansoeke word ingewag van tweetalige geregistreerde geneeshere vir die betrekking van Assistent-Sekretaris van die Mediese Vereniging van Suid-Afrika. Alhoewel dit van die suksesvolle applikant vereis kan word om in die Transvaal te werk, sal hy waarskynlik ver wag word om voorlopig 'n sekere tydperk by die Hoofkantoor van die Vereniging te Kaapstad deur te bring.

Die salaris aan die pos verbonde is op die skaal £1,250×50—1,750, plus 'n duurtetoelag van £352 vir getroude mans en £176 16s. 0d. vir ongetroude persone. (£100 van hierdie toelag sal vir pensioendoelindes by die salaris gekonsolideer word.) Die aanvangskersal volgens ondervinding wees en sal deur die Federale Raad bepaal word. Van die suksesvolle applikant sal verlang word om by die Vereniging se pensioenfonds aan te sluit.

Aansoeke vergesel van getuigskrifte en 'n gesondheidsertifikaat moet die ondergetekende vóór of op 31 Julie 1956 bereik.

Mediese Huis  
Waalstraat 35  
Kaapstad  
19 Mei 1956

A. H. Tonkin  
Sekretaris

# THE BENEVOLENT FUND : DIE LIEFDADIGHEIDSFONDS

The following contributions to the Benevolent Fund during April, May and June 1956, are gratefully acknowledged.

## Votive Cards in Memory of:

Rev. E. G. Malherbe *by* Dr. Vernon Brink.  
 Dr. C. A. Coates *by* Dr. and Mrs. R. D. Nelson.  
 Dr. E. J. Hamlin *by* Dr. A. J. Orenstein.  
 Mr. F. H. Sergeant *by* Dr. D. P. Marais.  
 Mr. C. C. Horn *by* Dr. L. L. Alexander.  
 Sister Glynne *by* Dr. L. L. Alexander.  
 Dr. R. Burns *by* Medical Graduates Association.  
 Dr. G. Delaponte *by* Dr. D. P. Marais.  
 Dr. Anton ter Beek *by* Dr. R. Geerling.  
 Dr. J. S. Palm *by* Dr. W. H. Opie.  
 Dr. J. M. Coplans *by* Dr. A. W. Sichel.  
 Dr. J. C. Gie *by* Dr. A. W. Sichel, Rex and Thora Cutler and Miss Boulton, Dr. and Mrs. E. C. Keet, Mr. Cecil Pereira, Dr. Robert Wolff, Mrs. A. M. Payne and Ruth, Dr. and Mrs. P. W. J. Keet, Dr. A. J. Ballantine, Mr. P. Stanley James, Mrs. G. J. Papst, Mr. and Mrs. Otto Papst, Mrs. P. S. Duffett and Daughter, Mr. Wm. C. Foster and Lucie, Mr. Hugo Andersson, Dr. J. J. Jacobson, Dr. and Mrs. D. S. Davies, Dr. F. P. Bester, Dr. Ivor Verster, Mr. and Mrs. G. J. Thomson, Lt.-Col. and Mrs. B. M. Woodhead, Mrs. E. Smuts and Miss E. van Brakel, Mr. C. S. Solomon, Dr. and Mrs. Hugo le Roux, Mrs. P. R. Hofmeyer,

Mr. W. F. R. Schreiner, Mrs. R. L. Scott, Sister Fraser Grant, Pamela and Rene, Mrs. D. Davis and Miss M. Pringle, Dr. A. J. Goldberg, Mrs. G. M. Marshbank.

Mr. Percy Granger *by* Dr. Vernon Brink.

Total Amount received from Votive Cards £55 11s. 0d.

## Services Rendered to:

Wife of Dr. W. F. Skaife *by* Drs. Grieve, Pein and Owendale.  
 Mrs. A. M. le Roux *by* Dr. Trotter.  
 Wife of Dr. W. Gilbert *by* Drs. Meyer, Jacobson and Dr. van der Burgh.  
 Dr. W. Jacob *by* Mr. J. G. Bickerton and Anaesthetic Clinic, Durban.  
 Daughter of Dr. J. A. Cowlin *by* Drs. Goldman, Shore and Abelson.  
 Mrs. L. A. Albertyn *by* Dr. G. V. Doherty.  
 Mrs. J. E. Cosnett *by* Mr. Harold Renton.  
 Mrs. L. J. Schewitz *by* Drs. Sims, Gluckman, Bloomberg and Lewin.  
 Dr. D. H. R. Vollett *by* Dr. T. Edmunds.  
 The late Mother of Dr. P. Doctor *by* Mr. Lunz, Drs. Maresky, O'Donovan and Strasburg.  
 Dr. W. Gilbert *by* Dr. C. W. Coplans.

Total Amount received from Services Rendered, £49 13s. 0d.

## Donations

£ s. d.	£ s. d.	£ s. d.	£ s. d.
Cape Western Branch	Donations from the	Dr. A. H. Sader ..	Dr. W. J. Naude ..
Members Collection	funds of the Medical	Dr. H. Saacks ..	Dr. A. G. Blyth ..
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hurst .. 3 3 0	Dr. R. J. McMahon	0 10 6	Dr. J. L. Parker ..
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petition .. 9 0 0	Dr. R. W. Nash ..	0 10 6	
Proceeds of a Dinner	Dr. A. H. Baxter ..	0 10 6	
Dance arranged by	Dr. F. C. Steyn ..	0 10 6	
Medical Wives As-	Dr. E. T. Dietrich ..	1 1 0	
sociation .. 155 6 8	Dr. J. A. van Zyl ..	0 10 6	

## PATHOLOGICAL LABORATORY SERVICES PROVIDED BY THE UNION HEALTH DEPARTMENT

### RULES FOR THE CARRYING OUT OF LABORATORY TESTS

In an extract from Government Notice No. 1073 of 22 June 1956,\* part of Rule 3 was inadvertently omitted. Rule 3 is printed in full below:

3. LABORATORY SERVICES WHICH ARE PROVIDED FREE OF CHARGE  
 The following services will be provided free of charge:

(1) Services in respect of the Diagnosis and Control of Infectious and Communicable Diseases.

(a) These free services will be restricted to laboratory tests for the diagnosis and public health control of the following diseases:

Anthrax,  
 bacillary dysentery,  
 bacterial food poisoning,  
 bilharziasis,  
 brucellosis,  
 meningococcal meningitis and septicaemia,

diphtheria,  
 enteric fever (typhoid and paratyphoid fevers),  
 leprosy,  
 malaria,  
 plague,  
 poliomyelitis,  
 puerperal fever,  
 rabies,  
 rickettsioses (louse and flea borne and tick typhus fevers),  
 scarlet fever,  
 smallpox,  
 tetanus,  
 tuberculosis,  
 venereal disease (gonorrhoea, syphilis and other venereal infections),  
 virus encephalitis,  
 and such other infectious or communicable diseases for which the Secretary for Health may consider it necessary, in the interests

\* S. Afr. Med. J. (1956), 30, 665.

of public health, to provide free laboratory services in a part or the whole of the Union for a specified or an indefinite period.

(b) These free services, as provided to registered medical practitioners in respect of their private patients, will be restricted to such laboratory tests as may be reasonably required to assist them in confirming the diagnosis in patients who are suspected, on sound clinical evidence, to be suffering from any of the diseases listed under para (a) above.

(c) These free services, as provided to local authorities will be in respect of out-patient clinic, hospital, domiciliary medical and sanitary services, which are established by them, under the Public Health Act, No. 36 of 1919, as amended, for the control of infectious and communicable diseases. These services will include all such laboratory tests as may be reasonably required for the diagnosis and the treatment of patients who are suffering, or suspected to be suffering, from the diseases listed under para (a) above, for ascertaining when such patients have become free of infection and for the public health control of these diseases including the detection of human 'carriers' of these diseases and the tracing of outbreaks of these diseases to their source with the object of preventing further spread.

#### (ii) Services in respect of the Diagnoses of Neonatal Haemolytic Disease.

These services will be provided free to registered medical practitioners, in respect of their private patients, to health centres and ante-natal clinics. The services will be restricted to such blood tests as may be reasonably required to determine whether a pregnancy is likely to terminate in acute haemolytic disease of the infant as caused by maternal sensitisation to blood antigens.

#### (iii) Services in respect of Detached Out-patient Clinics.

Free laboratory services which will include such tests as may be reasonably required for the diagnosis and treatment of disease in patients who are attending detached out-patients' clinics which have been established under section seventeen of the Public Health Amendment Act, No. 51 of 1946, as amended.

#### (iv) Services to Other Government Departments.

Such laboratory services as may be reasonably required by other Government departments, e.g. the Union Defence Force, the South African Police and the Prisons Service, in respect of free or subsidized medical services provided by them for their employees and the dependants of such employees.

### ROYAL COLLEGE OF OBSTETRICIANS AND GYNAECOLOGISTS

On 4 July 1956, at Durban, the first South African Regional Council of the Royal College of Obstetricians and Gynaecologists was inaugurated. The ceremony took place in the course of the Obstetrical and Gynaecological Congress which was being held in Durban. The inauguration was conducted by Mr. C. D. Read, F.R.C.S., F.R.A.C.P. & S., P.R.C.O.G., President of the Royal College of Obstetricians and Gynaecologists, who delivered an address, which will be published in a future number of this *Journal*.

Hitherto the affairs of the Royal College in South Africa have been managed by a Reference Committee appointed by the Council of the College in London. At the beginning of this year the Royal College ratified the formation of a South African Regional Council, following the precedent of Australia, Canada and New Zealand, in each of which Regional Councils have been formed during the last 10 years.

This is a highly significant development, indicating that in the eyes of the world of obstetrics and gynaecology South Africa has come of age, and takes its place as an equal with other countries. The Regional Council will have a large measure of authority in respect of obstetrical and gynaecological matters in the Union of South Africa. It has the right to advise and assist any body seeking its assistance, and can always call upon the support and wide experience of the Council of its parent body.

The following were elected to the first South African Regional Council.

*Fellows:* Prof. James Black, Johannesburg, Prof. E. C. Crichton, Cape Town, Dr. R. L. Impey, Cape Town, Prof. J. T. Louw, Cape Town, Dr. D. F. Standing, Durban, Prof. L. J. te Groen, Pretoria.

*Members:* Dr. F. N. Charnock, Cape Town, Dr. F. Daubenton, Johannesburg, Dr. H. Renton, Durban.

Professor James Black, the first Chairman of the South African Regional Council, was invested with the badge of office by the President. Other office-bearers are Dr. R. L. Impey, Vice-Chairman; Prof. J. T. Louw, Hon. Treasurer; and Dr. F. Daubenton, Hon. Secretary.

After the inauguration the President conducted the first admission ceremony to be held in South Africa, when two Members, Dr. J. O. E. Aphorpe and Dr. B. C. Murless, were elevated to the Fellowship of the College, and Dr. F. C. Wilkinson was admitted to the Membership.

There are today about 90 Fellows and Members of the Royal College of Obstetricians and Gynaecologists in the Union, besides a number of practitioners who hold the diploma in Obstetrics of the College.



Royal College of Obstetricians and Gynaecologists, South African Regional Council. *Standing (left to right)*—Dr. H. Renton, Prof. L. J. te Groen, Prof. E. C. Crichton, Dr. D. F. Standing, Dr. F. N. Charnock. *Sitting (left to right)*—Prof. J. T. Louw (Hon. Treasurer), Mr. C. D. Read (President), Prof. J. Black (Chairman), Dr. F. Daubenton (Hon. Secretary). *Absent*—Dr. R. L. Impey.

## PASSING EVENTS : IN DIE VERBYGAAN

Union Department of Health Bulletin. Report for the 7 days ended 28 June 1956.

Plague, Smallpox: Nil.

Typhus Fever. Cape Province. No further cases have been reported from the Queenstown district since the notification of 30 May, 1956. This area is now regarded as free from infection.

Epidemic Diseases in Other Countries.

Plague: Nil.

Cholera in Calcutta (India); Chalna, Chittagong, Dacca (Pakistan).

Smallpox in Kandahar (Afghanistan); Rangoon (Burma); Phnom-Pehn (Cambodia); Ahmedabad, Allahabad, Bombay, Calcutta, Delhi, Kanpur, Karikal, Madras, Pondicherry, Visakhapatnam (India); Dacca (Pakistan); Tourane (Viet-Nam); Nairobi (Kenya).

Typhus Fever in Baghdad (Iraq).

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Union Department of Health Bulletin. Report for the 7 days ended 5 July 1956.

Plague. Nil.

Smallpox. Cape Province. No further case has been reported from the Kenhardt district since the notification of 7 June 1956. This area may now be regarded as free from infection.

Typhus Fever. Cape Province. No further case has been reported from the Mount Frere district since the notification of 7 June 1956. This area may now be regarded as free from infection.

Epidemic Diseases in Other Countries.

Plague. Nil.

Cholera in Calcutta (India); Dacca (Pakistan).

Smallpox in Rangoon (Burma); Phnom-Pehn (Cambodia); Ahmedabad, Bombay, Calcutta, Cuddalore, Delhi, Kanpur, Karikal, Lucknow, Madras, Pondicherry, Visakhapatnam (India); Dacca (Pakistan); Suez (Egypt); Mombasa (Kenya).

Typhus Fever in Baghdad (Iraq); Alexandria, Cairo (Egypt).

\* \* \*

Mr. D. A. Muskat, Ch.M. (Rand), of 303-304 Pan-African House, corner Jeppe and Troye Streets, Johannesburg, has recently been awarded the Diploma in Medicine at the University of the Witwatersrand.

\* \* \*

A clinical meeting was held at the Workmen's Rehabilitation Centre, corner of Esselen and King George Streets, Hospital Hill, Johannesburg, on 6 July 1956 at 5 p.m. Similar meetings are to be held fortnightly at which cases will be presented by doctors who have patients at the centre falling within the scope of the Workmen's Compensation Act of 1941. These clinical meetings last approximately 1 hour, and a cordial invitation is extended to all practitioners to attend and take part in the discussions.

\* \* \*

Dr. J. E. Wolff of 310 Cavendish Chambers, corner of Jeppe and Kruis Streets, Johannesburg, is at present in Rio de Janeiro with Dr. A. Franceschetti, Professor of Ophthalmology, University of Geneva, Switzerland, visiting Dr. Nelson Maura, the eminent Brazilian ophthalmologist, in order to study his work.

\* \* \*

The John Ryle Memorial Prize. The Medical Association for the Prevention of War offers a prize of £75, under this name, for the best essay under the title 'A World Approach to Human Survival

and Health'. The prize is offered as a memorial to the late John Ryle, Professor of Social Medicine at Oxford. It is open to qualified medical practitioners and medical students of any nationality.

Entrants must first apply for particulars to the Secretary, Dr. D. L. Kerr, 291 Burntwood Lane, London, S.W. 17, in time to enable them to submit their essays by 31 December 1956, which is the last date on which essays will be received.

\* \* \*

A Centenary. The English firm of Smith and Nephew, whose South African 'offspring' is Smith and Nephew (Pty.) Ltd., manufacturers of Elastoplast products and Gypsona plaster-of-paris bandage at Pinetown, Natal, have recently celebrated their hundredth year of manufacturing. The South African company also became 25 years of age this June.

\* \* \*

The Twelfth International Congress on Occupational Health. The Permanent International Committee on Industrial Medicine having entrusted Finland with the organization of the 1957 congress, the organizing committee set up by the Finnish Government announces that the Twelfth International Congress on Occupational Health will be held in Helsinki on 1-6 July 1957. The support and attendance is invited, not only of doctors, but also of chemists interested in occupational medicine and hygiene, research workers and teachers in this field, engineers, social insurance workers, industrial and public health nurses, and others.

The Congress subjects will be Industrial noise; Evaluation of Invalidity; Industrial hygiene norms; and Cardiacs and work.

The section subjects are as follows: Human engineering; shift work and health; Industrial back; Applied psychology and occupational health; Occupational non-silicotic pneumopathies; Blood diseases in relation to industrial toxicology; Toxicology of new chemicals; Isotopes in toxicological research; Diseases due to stress and strain; Radiation—hazards and prevention; Training of industrial nurses; Health interviews in connection with physicians' examinations; Geriatrics and industrial nursing; The nursing aspects of rehabilitation in industry; Health education in industry; Industrial nurse with and without medical direction; How to meet the needs of small factories;

There will also be reviews of the following: Psychosomatic medicine considered from the aspect of social security; Recent advances in industrial ophthalmology; Control of chronic diseases in industry; Recent advances in silicosis research. A round Table Meeting will be held on Teamwork in occupational health. The official languages are English, French, German and Spanish.

A general social programme will be arranged, and a special programme for the ladies of the congress. Before and after the congress an opportunity will be afforded members to visit and acquaint themselves with the organization and operation of occupational medicine and hygiene in Finland and other Scandinavian countries. A film competition will be organized, and also an international technical exhibition.

The congress fee for members will be U.S. \$15.00, and for associate members \$10.00.

Communications in regard to contributions from members should be addressed to the Secretary General, Dr. Pentti Sumari, c/o Työterveyslaitos, Haartmaninkatu 1, Helsinki-Töölö. Notice of intention to be present must be made before 1 May 1957, after which date hotel accommodation cannot be guaranteed.

The South African members of the Permanent International Committee on Industrial Medicine are Maj. Gen. A. J. Orenstein and Dr. J. H. G. van Blommestein.

## BOOK REVIEWS : BOEKRESENSIES

## PSYCHOSOMATIC MEDICINE

*A Psychosomatic Approach to Medicine.* By Desmond O'Neill, M.D., M.R.C.P. (Lond.), D.P.M. (Eng.). Pp. 197 + vii. 25s. London: Pitman Medical Publishing Co., Ltd. 1955.

Contents: 1. Doctor and Patient. 2. Stress Disorder in Practice. 3. Physical Changes in Emotional States: Experimental Work. 4. Stress Symptoms. 5. Therapy of Stress Disorders. Appendix.

This is a valuable addition to the growing number of books and papers which stress the need for a holistic approach to the patient. Dr. O'Neill gives an easily-read outline of the history and theoretical bases of psychosomatic medicine. His clear account of the ways in which emotional stresses produce many common symptoms and syndromes is accompanied by useful references to the literature and the use and place of psychiatric methods in family practice are clearly presented. The author successfully shows

that, although searching for the emotional origins of illness demands more of the doctor, it yields greater satisfactions.

To those who have had psychiatric training or experience, some of the successes in the case histories may look too easy, and perhaps Dr. O'Neill has over-simplified the problems presented by the neurotic patient. Nevertheless, there are very few practising physicians, and certainly no senior medical students, who would not benefit from a study of this book.

H.T.P.

#### EXAMINATION QUESTIONS

*The M.B., B.S. Finals (1946-1954):* By Francis Mitchell-Heggs, T.D., M.B., B.S. (Lond.), F.R.C.S. (Eng.), F.R.C.S. (Edin). Fourth Edition. Pp. xxvi + 62. 10/6 net. London: J. & A. Churchill Ltd. 1955.

*Contents:* Extracts from Regulations of the University of London. Surgery: Clinical Problems. Local and Specific Infections. Injuries of Bones and Joints. Diseases of the Alimentary System. Diseases of the Ear, Nose, Throat and Eyes. Diseases of the Genito-urinary System. Orthopaedic Surgery. Miscellaneous.

Surgical Anatomy. Surgical Pathology. Obstetrics and Gynaecology: Normal Pregnancy. Abnormal Pregnancy. Abnormal Labour. The New-born Infant and Puerperium. Gynaecological Problems. Diseases of the Uterus, Ovary and Fallopian Tubes. Medicine: Clinical Problems. The Ductless Glands, Metabolism and Nutrition. The Cardio-vascular System. Conditions of the Blood. The Nervous System. The Respiratory System. The Alimentary System. The Urinary System. The Skin. Medical Infections and Fevers. Applied Pharmacology and Therapeutics: Clinical Problems. Specific Treatment. Symptomatic Treatment. Toxic Effects. Forensic Medicine: Toxicology. Causes of, and Investigation after, Death. Sex, Pregnancy and Infancy. Legal Terms and Procedure. Public Health: Vital Statistics. Infectious Diseases. Sanitation and Hygiene. Nutrition. Pathology: General Pathology. Laboratory Investigation. Bacteriology. The Alimentary System. The Cardio-vascular System. The Genito-urinary System. The Nervous System. The Respiratory System. The Glandular and Reticulo-endothelial System and the Blood. The Skeletal System. Practical Pathology.

As will be seen from the extensive list of contents above this little book covers the wide variety of questions set in the M.B., B.S. Finals of the University of London over the last 10 years. Since this book was first published in 1935 four editions have appeared, testifying to its usefulness to students and teachers.

A.H.T.

### CORRESPONDENCE : BRIEWERUBRIEK

#### OVERWORK

*To the Editor:* 'Overseas Daily Mirror' of 8 June 1956 contains (on page 6) news about electric-meter readers in London complaining that they have to read an average of 30 metres a day. Because of this complaint an investigation about the 'overwork' was conducted.

Has it occurred to anybody to mention the fact that practitioners in the National Health Service in Britain (and Benefit Society practice in South Africa) regularly attend to more than 30 cases daily and to many more if there is even a slight epidemic of measles or influenza? It is obviously impossible to attend to so many patients and pay full attention to them. It seems incomprehensible that those requiring medical services do not see it and do not demand a radical change in their own interest.

*No safety in numbers*

4 July 1956

#### THE HEROIN BAN

*To the Editor:* In your leading article<sup>1</sup> on heroin (16 June 1956) you suggest that one of the main reasons why this drug has not been banned from medicine in Britain is because it is considered to be valuable in therapeutics. This is only partially true. The heroin controversy in Britain is exceedingly confusing to anyone who did not follow day-to-day developments at the time, because of the vast amount of uninformed comment, much of which came, sad to say, from the medical profession itself.

A very important point, which you do not mention, is that there has never been a shred of evidence that heroin legally manufactured in Britain has contributed to the international contraband traffic. The heroin made in Britain is all accounted for under an effective control system, and cessation of production would not deprive addicts abroad of their supplies because these are produced by secret manufacture from contraband opium and morphine—an easy procedure. It is the effective international control of opium and morphine traffic which will prevent heroin from getting into illicit markets, not the suppression of a manufacture which has never contributed to the problem in any way.

Authority for all these statements can be found in the United Nations publication 'Bulletin on Narcotics'. The futility of a ban on legal manufacture is shown by these figures: Britain consumes 55% of the legal world-supply of heroin, and has 54 addicts; the USA consumes no legal heroin and has 60,000 addicts (at least). The British Government was repeatedly challenged to justify its proposed ban. The British Medical Association put these questions: 'Has the Government any evidence that heroin from officially controlled sources in this country has been getting into the illicit trade? Has the Government any other evidence that a ban on the manufacture of heroin in Britain will help to reduce heroin addiction abroad? If the Government cannot produce such evidence, will it state its real reasons for persisting with this ban?' There was no reply.

Lord Jowitt, in the debate on the heroin ban in the House of Lords on 13 December 1955, said: 'We want from the Government . . . some explanation as to how it is that banning the production of heroin in this country will assist the problem in the United States'. (The USA is the principal instigator of the proposal to ban legal manufacture.) 'You must establish something more substantial than a mere gesture of international solidarity'. There was no reply.

It was then, as now, difficult to resist the conclusion that an ill-informed decision has been reached and that it was being persisted in for reasons unrelated to the merits of the case. There can be little doubt that the British medical profession would be willing to discard heroin if anyone, including the United Nations Commission on Narcotic Drugs, could produce the smallest evidence that the grave social problem of heroin addiction in other countries would be benefited, but until such evidence is produced they rightly feel, with Professor G. Brouet of Paris, that 'if morphine continues in current use, heroin is so easy to prepare that there is little point in banning it internationally. If there was any absolute need to prohibit its use in therapeutics in order to combat addiction, the principle might conceivably be accepted; but if the effect of such prohibition were to be illusory, as we fear it would, it is hard to see why patients who may benefit from it need be deprived of it'.

The conclusion to the furore came when Lord Jowitt threw doubt on the legality of the Government's action, and the ban was withdrawn. It seems that a Parliamentary Bill would be required to ban heroin. Let it be hoped that the British Government will allow the matter to rest unless or until evidence is found that heroin legally made in Britain is being misused. It is common ground that it would be wise to ban heroin exports and this has been done.

The control of heroin addiction surely lies in suppression of known sources and not in suppression of supplies for proper medical use. Thus the clinical utility of the drug is a secondary matter which will only become relevant if it should ever appear that suppression of legal manufacture in Britain would contribute to the international problem.

It is even possible to throw considerable doubt on your observation that heroin is 'the most dangerous of all drugs of addiction' but unfortunately space does not permit me to attempt this exercise; so may I conclude by drawing your attention to the fact that it is reported from Finland that heroin addicts are spontaneously changing over to methadone, one of the recommended 'safe' substitutes for heroin.

D. R. Lawrence

Durban Medical School  
Umbilo Road  
Durban  
2 July 1956

Department of Medicine, University of Natal, and University College Hospital Medical School, London

1. Editorial (1956): S. Afr. Med. J., 556.